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Report of the PAX Company Arsenic Advisory Committee to the Environmental Protection Agency

PAX Co Arsenic Advisory Committee, Schenectady, N Y

Prepared for

Environmental Protection Agency, Washington, D C

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REPORT

of the

PAX COMPANY ARSENIC ADVISORY COMMITTEE

to the

ENVIRONMENTAL PROTECTION AGENCY

Submitted, May 13, 1973

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The purpose of this report is to assist the EPA Administrator to assess the safety in use of the PAX product and of related arsenicals. As a result of the study, there was no unanimity in the attitudes toward the safety of PAX, but the Committee was unanimous in urging further research. The pesticide in question is the PAX Company Three-Year Crabgrass Control product containing 25.11% arsenic trioxide, 8.25% lead arsenate and 4% nitrogen as ammonium sulfate.

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May 12, 1973

Mr. William D. Ruckelshaus
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Washington, D.C. 20460

Dear Mr. Ruckelshaus:

As chairman of the PAX Company Arsenic Advisory Committee, I am privileged to submit our report. We trust this will help you to assess the safety in use of the PAX product and of related arsenicals. Although, as one might expect, there is not unanimity as regards attitudes towards the safety of PAX, the Committee seems unanimous in urging further research.

Your Committee was impressed with the aid accorded us by Clayton Bushong and Charles Lewis. The arsenical literature is vast. We could not cover it in the time allotted, but great efforts were made to enable us to do so.

In about 40 years study of the arsenical problem, I have become convinced that overreactions to arsenophobia, plus the inability to relate the basic importance of As to life, has led to many unscientific regulations. It has negated the safe uses of arsenicals in medicine. Anti-arsenic regulations vary greatly between countries. But all reflect the basic mistrust of this versatile and useful element. As noted elsewhere, we tend to misjudge the toxic trace elements. One can only hope that, to correct such misjudgments, your agency will recognize the futility of laws and regulations which run counter to biology itself. If this is accomplished, and followed by purposeful research to clarify the beneficial roles of trace elements, agriculture, medicine and the environment will surely benefit. Further repression of safe uses of the toxic trace elements will be tolerated for a time, but is inconsistant with Nature and must lead to eventual failure. The pattern seems clear in the confusion shown by governments regarding the safe uses of selenium and mercury in agriculture.

These are semantic problems, best understood in light of Korzibsky's Science and Sanity, and Wendell Johnson's People in Quandaries. Both point out that the only way man can solve controversial problems is to depend on objective interpretation via the scientific method, i.e. repeatable experiments. Sufficient has been done with PAX to warrant that, except for avoidable accidents, the product can be used safely. The crux of the problem appears in the letter to the Committee by Drs. Kearney and Woolson.

Respectfully submitted,

D V Frost

Douglas V. Frost, Chairman
PAX Company Arsenic Advisory Committee

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REPORT OF THE PAX COMPANY ARSENIC ADVISORY COMMITTEE

The Committee is charged to investigate and to weigh risks versus benefits of the PAX Company Three-Year Crabgrass Control product containing 25.11 % arsenic trioxide, 8.25 % lead arsenate and 4 % nitrogen as ammonium sulfate. Under Interpretation 25 of the Federal Insecticide, Fungicide, and Rodenticide Act, products for use in and about the home shall contain no more than 1.5 % arsenic trioxide or 2 % sodium arsenite. On the basis of evidence presented by Done and Peart (1), the United States District Court for the District of Utah (Central Division), held that PAX Three-Year Crabgrass Control had not been shown hazardous to humans and that Interpretation 25 was inappropriate. Despite the reassuring evidence by Done and Peart that the PAX Company Crabgrass Control product did not present a hazard to humans, reports appeared by William B. Buck (2), veterinary toxicologist at Iowa State University, clearly establishing that grass clippings from PAX-treated lawns might be lethal to animals. Misunderstandings stemmed from 1) the efficacy of PAX depends on action at the soil surface to inhibit germination of crabgrass and growth of the annual blue grass, Poa annua. It is not absorbed to any extent by grass, but may be picked up, particularly by rotary mowers, along with grass clippings. 2) as now stated on the PAX labeling, (but not always so stated in the past), PAX-treated grass clippings should not be fed to animals.

Whereas PAX is used in Utah and other western states for turf improvement on golf courses, somewhat similarily to calcium arsenate and lead arsenate, the primary sale is for use on home lawns. Variations of PAX, all based on combinations of arsenic trioxide and lead arsenate, have been marketed for about 20 years for home use. This use is covered under a U.S. Patent (3). Beginning in 1970, various states enacted laws requiring the licensing of qualified applicators for the use of toxic pesticides. Because arsenicals are widely known for their toxicity, their use on home lawns was banned in effect in many states by such licensing requirements.

The Federal Environmental Pesticide Control Act of 1972 (Public Law 92-516) may in time require that each state establish regulations for certified application of hazardous pesticides. Under this law, each state would regulate the sale and use of pesticides within state boundaries. Such action would eliminate the right of individuals to use many materials used safely, with few exceptions, for decades.

The safe uses of arsenicals have challenged science and man's ingenuity throughout recorded history. Although virtually taboo now for use in medicine, inorganic arsenicals were among the first medications and certain organic arsenicals were medicine's first "magic bullets" (cure for veneral disease). Inorganic arsenicals were man's first economic poisons and first pesticides. The uses of inorganic and organic arsenic in agriculture are too varied and complex to discuss here (4,5,6). Suffice it to say that science still has much to learn of the benefits to be derived from this amazing but controversial element. Clearly, inorganic arsenicals, such as PAX, exert beneficial effects on turf. The advantages of such effects can, however, only be realized by consideration of the fact that misuses of the product can represent definite hazards to animals. Such hazards are avoidable if the product of PAX-treated grass clipping is not fed.

The ability to employ compounds of toxic trace elements such as arsenic to man's advantage is one of the great challenges of our times. Available sources of phosphate, the limiting ingredient of fertilizers, are being rapidly depleted. How arsenicals influence phosphorylations in plants and animals is a major scientific question. Evidence seems clear that the benefits from arsenical use in obtaining good turf depend in part on the avoidance of added phosphate. Whether and to what extent arsenic may substitute for phosphate in plants, particularly in turf grasses,deserves consideration as an economic measure in phosphate conservation. The extended (three year) turf improvement effects claimed for PAX, verified by unsolicited testimonials (Appendix 1), also warrant economic consideration in the assessment of risks vs. benefits. The value of arsenicals to stimulate nitrogen fixation, thus sparing nitrogen fertilization of turf is a further economic consideration (40,40a).

Section 1. Chemistry and Arsenic Cycle - D.V. Frost

Milestones in the chemistry of arsenic have been prominent in the history of both chemistry and biology. Greater emphasis has been placed on adverse effects and suppositions of such adverse effects than on the good faces of arsenic. To place the situation in perspective, it seems appropriate to outline many of the milestones in As history. Clearly, there has been and still remains much subjectivity in how man veiws matters relating to "arsenic".

Milestones

2000 BC	Arsenicum, arsenik (As_2O_3). The sublimate from copper and iron ores.
400 BC	Hippocrates used arsenic sulfide salves to treat ulcers.
1250 AD	Elemental As prepared by Albertus Magnus.
Middle Ages	As_2O_3 used by professional poisoners. Laws finally passed against such uses.
1775	Scheele volatized arsine, AsH_3; a way to separate As from most other elements.
1786	Fowler's solution, 1% As_2O_3, as potassium arsenite. Still said in 1912 to be the best medicinal in the pharmacopoeia.
1820	Arsenic cancer myth begins in writings of J.A. Paris
1836	Marsh test for As, via Scheele's arsine.
1839	Gmelin wrote of volatile arsines produced by molds.
1842	Cacodyl, $(CH_3)_2As-As(CH_3)_2$, first organic arsenical. Coincided with Wohler's synthesis of urea.
1868	First pesticide-copper acetoarsenite-controlled Colorado potato beetle. Shades of the Irish potato famine, 20 years before.
1887	Hutchinson's idea: Fowler's solution → hyperkeratosis → skin cancer. The idea grew by reiteration into medical tradition. Industrial exposure to As_2O_3 fails to cause cancer.
1900-3	Royal Commission on Arsenical Poisoning established world's first tolerance, unofficial, and without scientific basis, but ending up with force in courts of law.
1910	Chemotherapy began with Ehrlich's magic bullet, arsphenamine.
1938	Moxon found arsenicals can counteract selenium toxicity.
1942-3	Kennaway-Hueper polemic in Lancet re Hueper's allegations against As based on J.A. Paris' writings, 1820-2.
1946	Organic arsenical feed additives control diseases in poultry and swine, stimulate growth and improve feed efficiency.
1956	Arsenic acid emerges as a desiccant for cotton; cacodylates and methanearsonates as desiccants, defoliants, herbicides.
1958	Arsenical feed additives impugned under Delaney clause.

1959	France bans organic arsenicals, antimonials, and estrogens from use in poultry feeds. EEC countries follow suit. England legalizes an Arsenic in Foods Regulation, its first official tolerance.
1962	France bars import of poultry from countries where arsenicals are permitted; exempts poultry livers used in French pate.
1963-7	Food and Drug Administration clears four organic arsenical feed additives of carcinogen stigma. Arsenic acid permitted as desiccant for cotton.
1968	AAAS-DOD polemic on defoliants and herbicides in Viet Nam.
1969	Safety of PAX upheld in Utah District Court. Fallon water incident. Accidental poisoning of horses fed PAX-treated grass clippings in Iowa. Dr Buck confirmed possibility at Iowa State.
1970	New York classed arsenicals as residual pesticides requiring licensed use. Cacodylic acid use in Viet Nam stopped by executive order.
1971	Licensed uses of tricalcium and lead arsenates on golf courses achieved acceptance in most states.
1972-3	Retrospective study of orchardists exposed to lead arsenate in 1930s and '40s revealed no adverse health effects, possibly improved health.

Arsenic trioxide

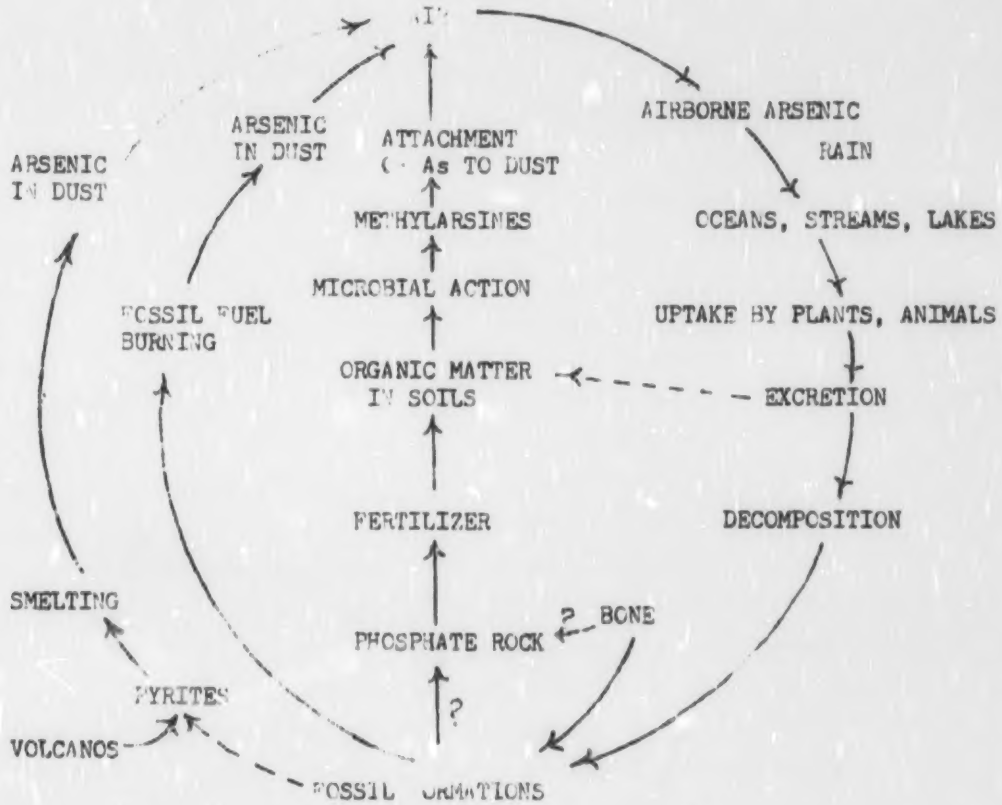
The form in which arsenic first became available was as the sublimate (or distillate) from metallic ores. "White arsenic" or simply arsenic as it was known for centuries, is the oxide of the element. It is only sparingly soluble in water and is non-ionic. In granular form, it is only about one tenth as toxic as when dissolved in alkali to form a water-soluble salt, such as sodium arsenite. Failure to understand the chemistry and biochemistry of As_2O_3 appears in the definition of Fowler's solution as a 1 % solution of arsenic trioxide. When arsenic trioxide is dissolved in water, it forms arsenious acid. When the solution is evaporated, all water is lost, forming again the anhydide As_2O_3. It becomes available biologically only if dissolved to yield arsenous acid. The rate at which As_2O_3 dissolves in the tract of animals varies with particle size and between species. This is developed in the report by Done and Peart (1). Interpretation 25 assigned greater toxicity to arsenic trioxide than it did to sodium arsenite. This is in retrospect an obvious error. But the tendency to view arsenic trioxide as comparable to sodium arsenite is so firmly grained in the literature that only conscious effort and reeducation can set the record straight.

Many inorganic arsenicals in soils are far less soluble than arsenic trioxide. As_2O_3 is probably not a common constituent of soil, being converted to even less soluble forms by chemical reaction. Evidence suggests that arsenic may enter into active global cycling and that As_2O_3 in dust may be an important part of this cycle. Ferguson and Gavis (7), in an excellent review of the arsenic cycle in natural waters, stated that "There is no evidence that man is likely to change world-wide distribution of arsenic appreciably...We must conclude that present knowledge of the pathways of the arsenic cycle is inadequate to allow good management...". That concentrations of arsenic represent a public health hazard appears valid only for the unique situation in which very large amounts of As_2O_3 are permitted to escape from smelters. Harkins and Swain (8,9) detailed one such occurrence early in the century; Birmingham (10) and others (10a) more recently. The fact that water supplies can become contaminated with toxic levels of arsenic, again around smelters, was described by Borgono and Greiber (11).

Occurrence of high levels of arsenic in natural waters was reported by Grimmett et el.(12). This study of the possible relation of arsenic in soils and waters in a geothermal area of New Zealand deserves special consideration and calls for more research. The study showed that remarkably high levels of bound forms of arsenic, such as arsenic sulfide in muds, were non-toxic to cattle. On the other hand, the experimental feeding of sodium arsenite to a heifer at a relatively low level of arsenic proved to be lethal. Working in the same area, Lancaster et al.(13) reported that 288 ppm of arsenic in lake weed proved entirely non-toxic when fed at 20 % of the daily ration for sheep. The biochemical data suggests that the arsenic in the lake weed was in bound form and was less toxic than more available forms of arsenic might have been. As is true for many other species, there is little agreement on the toxicity comparisons for arsenite in sheep. For instance, Clarke and Clarke (14) reported that as little as 2.6 mg As as sodium arsenite per kg of body weight proved lethal to sheep On the other hand, Bucy et al. (15) reported no toxic effects for 56 days to lambs fed 9.1 mg As per kg per day as potassium arsenite. If nothing else, such comparisons serve to show how variable the toxicity of even the most toxic forms of arsenic may prove to be among investigators. The Lancaster study raises further questions about those combined forms of arsenic which occur in the arsenic cycle.

The key role of microorganisms to release undue concentrations of arsenic from soils is part of the arsenic cycle depicted by Frost (6). In view of advancing knowledge, the *As* cycle can now be shown as follows:

ARSENIC ECODIAGRAM - D.V. Frost


AIR
AIRBORNE ARSENIC
RAIN
OCEANS, STREAMS, LAKES
UPTAKE BY PLANTS, ANIMALS
EXCRETION
DECOMPOSITION
BONE
?
PHOSPHATE ROCK
?
FOSSIL FORMATIONS
VOLCANOS
PYRITES
SMELTING
ARSENIC IN DUST
FOSSIL FUEL BURNING
ARSENIC IN DUST
ATTACHMENT OF As TO DUST
METHYLARSINES
MICROBIAL ACTION
ORGANIC MATTER IN SOILS
FERTILIZER

Because the arsenate is readily precipitated by metal hydroxides, only about 0.003-0.02 ppm of As is found in most natural waters. Nevertheless, aquatic plants transmit As at relatively high levels to fish and sea foods as part of the food chain. Land animals contain lower levels of As on average, again as part of the food chain.

Goldschmidt (16) in his classical review of the geochemistry of arsenic noted that forest humus contains up to 300 ppm of arsenic. He hypothesized that over eons of time, this led to the high concentrations of As in fossil fuels. Coal, metal ores, and phosphorites all contain appreciable levels of As, strongly suggesting that As is associated with biology and with elements essential to life. The burning of fossil fuels releases large amounts of As to the atmosphere. Whether this is more or less than the amounts released by biological mechanisms is not known. Although quantitative estimations are difficult, it has been estimated that about 29 x 10^4 tons of arsenic were released via the burning of coal from 1900 to 1971. The estimated 50,000 metric tons of arsenic produced commercially each year calculates to be about 10 times the release of As from the burning of coal. Sulfite ores, predominately arsenopyrite (FeAsS), and the various non-ferrous ores from which commercial arsenic is obtained, impart appreciable levels of As_2O_3 to the atmosphere through smelting.

Unless the arsenic is artificially removed, phosphate fertilizers impart appreciable levels of arsenic to soils. Superphosphates made with sulfuric acid also containing As as impurities, may contain up to 0.14 % As (25). The benefit possible from a relatively high level of As in soils is not well documented.

According to Williams and Whetstone (17), soils from various parts of the United States and Mexico contained from .3 to 40 ppm As. Olson et al. (18) reported 7-18.4 ppm of arsenic in South Dakota soils with plants containing from 1.2 to 4.3 ppm of As. In only a few cases did the soils studied have more than 3 ppm As. Except under very artificial conditions, such as the poisoning of soils by lead arsenate spray, or the occurrence of high levels of bound As in geothermal areas (12), high concentrations of As do not appear to be a problem in soils. Arsenophilic molds, fungi, and bacteria are stimulated by unusual concentrations of arsenic in most of its forms and are known to release methylated arsines to the atmosphere from soils of high As content (16).

The role played by As_2O_3 as an aerosol particle in the arsenic cycle appears from many considerations to be significant. A remarkably high level of As in city dusts, averaging above 5 ppm, was reported by various authors (19,20,21). In a careful review of the air pollution aspects of arsenic and its compounds, prepared in 1969 for the National Air Pollution Control Association, Sullivan and others (22) provided the most complete treatise so far available on levels of As in city vs. urban air. Other than the well-defined arsenical pollution problems which have occurred in and around smelters, no evidence of adverse effects from arsenical air pollution were reported. Although skin abnormalities in humans were reported, particularly among arsenical workers (10,10a), no adverse systemic illnesses in humans have been clearly related to exposure to arsenicals. For purposes of this review, attention may be called to the very recent disclosure by W.C. Nelson et al. (23). This retrospective epidemiologic study of orchard workers exposed to lead arsenate spray around Wenatchee in the 1930s and '40s indicated that those exposed had, if anything, a somewhat better health record than was true for people of similar age not so exposed.

In a report of various trace elements in the atmospheric environment, Peirson et al. (24) note that the air concentration of As is far less in rural than in industrial areas This study again points the need for aerosol identification of the form or way in which As is transported through the atmosphere. The level of arsenic was reported to be far less than that of the macroelements, Na, Al, Cl, and Ca. The As level was reported well below levels of Fe, Zn, Pb, Cu, Mn, Ni, V, and Br, - about comparable to the level of Cr, but more than that of Se, Co, or Hg. The rate at which various trace elements return to earth in rain or as dust was also considered It appears from this study that most elements are cyclical in nature. The critical question remains as to what levels are beneficial and what levels harmful for each element.

An excellent study by Tremearne and Jacob (25) on the arsenic in natural phosphates and phosphate fertilizers, revealed a range of 0.4 to 188 ppm As in mineral phosphates. Whether this association results from bioconcentration of As with phosphorus is still not clear. The following conclusion was drawn: "When all the factors affecting the action of arsenic on plants and on soil organisms under practical conditions of farming are taken into consideration, it seems very unlikely that the quantities of arsenic contributed to the soil in phosphate fertilizers are sufficient

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to produce toxic effects even with very large annual applications of the fertilizer over extended periods of time.". Despite this USDA Technical Bulletin of 1941, the British government succumbed to arsenophobia in the late 1950s to the point of requiring that superphosphates made in England be so treated as to contain relatively little arsenic. As noted elsewhere (26,27), this action resulted in the inadvertant removal of selenium as well as *As*. This led in turn to an epidemic of selenium deficiency in many species of livestock in New Zealand.

In the United States, the chronic fear of arsenic led to governmental action in the early '60s to reduce the arsenic limit in food phosphates from 10 to 3 ppm.(6). Subsequent studies showed (28,28a) that representative foods in the U.S. contain quite low levels of arsenic, generally well below existing tolerances. Indeed, one may wonder if the level of arsenic in human foods has not been reduced too much. The fact that certain arsonic acids have been shown to improve the well being, feed efficiency, and reproductive performance of turkeys, chickens, and pigs, raises the question whether arsenicals may have an as yet unrecognized nutritional value for humans, as well as for animals. Morrison (29) established experimentally that the arsenic content from kitter in poultry houses did not appreciably increase the arsenic content of soils or crops when used as a fertilizer.

Various other reports have indicated that *As* is not cumulative in the human food chain. This can be explained by 1) the fact that *As* is only sparingly absorbed by plants, 2) the apparent fact that *As* in plants is not readily available to animals, and 3) that microorganisms to a very large degree prevent the accumulation of *As* in soils, making for a continuing cycle of arsenic via water, land, and the atmosphere.

Need for Historic Perspective for Safe Arsenical Uses

Laws, regulations, and medical taboos have repeatedly gotten ahead of science regarding the safe uses of arsenicals. Confusion between the toxicity of As and Se first led to an association between "arsenic" and "cancer", later to placing sole blame for the British Beer Poisoning Epidemic of 1900 on As (6). This in turn led to the tolerance setting for all types of arsenical residues, even for traces of arsenic in food phosphates (26). There are many indications that arsenicals play a positive role in the nutrition of plants and animals Thus, the tendency to avoid as much arsenic as possible may in time prove harmful to health.

For the purpose of this review, it seems significant to note that the diverse roles of As in biology involve its oligodynamic potential to act as a catalyst in trace amounts but as a toxin at higher levels. These effects of lead arsenate and other arsenicals were well described in the early 1900s. For instance, Haywood and McConnell of the U.S. Dept. of Agriculture (37) noted early in this century "in very minute quantities, arsenic appears to exert a stimulating effect or act as a tonic (on foliage) as it does on animals. It is probably this action which, by accelerating the functional activity of the leaf and producing more rapid assimilation, causes the excessive reddening and hastens the maturity of the fruit. On the other hand, if too large an amount is absorbed, it has a toxic effect, resulting in retarded assimilation, which in turn will cause the fruit to shrivel and drop before it has matured." This refers to the use of lead arsenate in apples The use of lead arsenate to accelerate the maturation and sweetening of citrus fruits must also reflect stimulation of plant metabolism by traces of arsenic (38). This effect, although still not well understood, was first reported before 1900 and is of value in the economy and timing of citrus production. Stewart and Smith (39) reported visible stimulation of plant growth, particularly root growth, for peas, radishes, beets, potatoes, and beans, with traces of arsenates. They concluded "...that the accumulation of arsenic in soil, as the result of spraying of orchards if not continued to excess, may be beneficial rather than injurious".

Extensive studies by Greaves (40) on the relation of arsenic to nitrogen fixation by soils indicated that the nitrification was greatest when the water soluble arsenic content of the soils was about 10 parts per million. Greaves noted that sodium arsenite became toxic to plants at a concentration of 40 ppm but that lead arsenate was not toxic and continued to stimulate nitrogen fixation by soil Azotobacter even at 400 ppm. The optimal level of 10 ppm As conforms to that of Woolson et al. (36) regarding the effect of As level on growth of corn. Greaves, who worked at the Utah Experiment Station, also noted the ability of arsenate ions to increase the bioavailability of phosphate ions to plants, a most significant observation in light of the phosphate resource limitation facing agriculture.

The remarkable versatility of arsenic is thus glimpsed in evidence that it has something to do with both phosphorylation and with nitrogen metabolism. As early as 1897, Stoklasa (41) had reported that, although arsenic acid could not replace phosphoric acid in the living cell, it did appear to substitute in part by stimulating plant growth. In 1901, Stutzer (42) raised the question whether the arsenic naturally present in superphosphates, i.e. up to 0.1 % could be harmful. He concluded after experimentation that it would be harmless to soils, plants, or consumers of foods grown on such soils. The irrational fear of "arsenic" led more than 50 years later to removal of As from superphosphates made in England. This led in turn to a tragic loss of livestock in New Zealand from selenium deficiency. Dr. John Barnes of the British Medical Research Council wrote me in private communication that this need not have occurred (26). It is precisely this type of overreaction to the safe uses of arsenicals that one may hope to avoid here. Such can be accomplished best by further research and by public recognition and appreciation of the benefits to be derived from safe uses of the toxic trace elements.

A level of about 14 ppm of arsenic was reported in the National Bureau of Standards Standard Reference Material, orchard leaves. This was a composite sample from various fruit trees in which leaves from arsenical-sprayed trees were excluded. Again, trying to learn from history, one finds in early work by Jadin and Astruc (42), the statement "The chlorophyll-bearing parts of plants always give a higher arsenic content than any other portion." If As indeed stimulates photophosphorylation (31), this association with chlorophyll-bearing parts of plants and the stimulatory effects of As for turf grasses becomes more understandable.

The "catalytic fertilizer" effect at about 10-75 ppm of available As in soils, along with its toxicity to plants at higher levels, was thus known to agronomists at least 60 years ago. Prolonged soil sterilization with sodium arsenite and high levels of arsenic trioxide came about in the 1930s (32). Discovery that desirable grasses were improved by arsenic trioxide and lead arsenate at levels which suppressed weeds, undesirable grasses, grubs and worms, and that this effect extended for several years, again came about through careful and painstaking research (3,33). More recently, Liebig (44) provided criteria to diagnose the arsenic status of soils for control of arsenic toxicity. His extensive tables showed that little arsenic gets into the edible parts of plant products, with roots and leaves showing the highest levels. He stated "Apparently, the effect of arsenic toxicity is such that plant growth is limited before large amounts of arsenic are absorbed and translocated to the top."

In reviewing air pollutants which affect animals, recent studies by Lillie (45) found no reported adverse effects from arsenic inhalation. He noted that the phosphate levels were reduced below normal in tissues of arsenate-poisoned rabbits. This fits the hypothesis that excesses of arsenate ions interfere with phosphorylation, thus causing general metabolic toxicity. It is consistent with evidence that low levels of arsenate catalyze phosphorylation (6).

In a plea for reality in evaluation of environmental health problems, Stokinger (46) listed seven guidelines. He exemplified the need to avoid unnecessarily severe standards by relating the situation in Fallon, Nevada. Fallon city water has exceeded the PHS-WHO limit for 40 years, with no evidence of toxicity to the people who use the water. This is a striking demonstration of the unreality prevalent among those not aware of the entire situation. That the setting of unrealistic standards must make in time for failure of our society was argued by Stokinger.

In my view, ability to be scientifically right about elements must in time determine our capacity for survival among them. The need to balance risks versus hazards in this controversial situation and to arrive at a wise decision should go hand in hand with the encouragement of arsenical research. Full advantage may thus in time be realized for the diverse roles apparent for arsenicals in agriculture, turf improvement being only one of many.

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ARSENIC TRIOXIDE AND LEAD ARSENATE IN SOIL

with

Recommendations Regarding PAX 400 3yr. Crabgrass Control

Dr. Paul J. Zinke

Introduction

The ultimate decision regarding the relative safety of adding to soil a combination of lead arsenate and arsenic trioxide as used in the PAX 3 year Crabgrass Control depends upon several factors. Among these are: 1. The concentration of the material in the soil, 2. the relative solubility of the various arsenical compounds which it will form in the soil, 3. the possibility of the later formation of even more toxic compounds in the soil, and 4. the possible physical concentration which may occur by various processes of soil movement.

These factors will be discussed in this report and a recommendation made regarding the apparent hazards and possible methods of safe use of the product will be made.

PAX 400 As A Soil Additive

The main concern with the use of this material lies in its content of arsenic. An important aspect to be considered in evaluating the relative safety of the product is the amount of arsenic and its possible concentration as added to the soil. The material as added is 25% Arsenious Oxide (As_4O_6) and 8.25% arsenate of lead ($Pb_3(AsO_4)_2$). Assuming 82.4% As in the arsenious oxide, and 16.6% as in the lead arsenate the total arsenic content in the PAX 400 is estimated to be 22%.

When given the specified application rate of the material to the soil as 40 lbs. per 2000 square feet the rate of application on a square meter basis is 99.7 grams of PAX 400 containing 21.49 grams of elemental arsenic. This is approximately 191.26 pounds of elemental arsenic per acre.

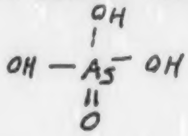
It is of interest to determine the concentration which this represents in the soil for comparison with other references on arsenic concentrations in soils. If the material added remains in the top centimeter of the soil having a bulk density of 1.0, the concentration of arsenic in the soil will be 2,100 ppm. Presumably this high concentration would occur only for a short time as instructions call for washing the material into the soil by intensive watering. Assuming that ultimately the material will be leached, (however, Stadtherr found less than 6% of it leached beyond ½" with 16" of applied water) and fixed or stored in the top 10 cm. of the soil the estimated concentration of arsenic in the soil a short time after application may drop to 210 ppm. This compares with natural background arsenic contents in soils that may range from 1-70 ppm (Arnott and Leaf), and thus could be considered to be a very high arsenic content for soil.

The persistence of this material in the soil and its release in quantities which may be toxic to plants, or concentrated by them in toxic quantities depends upon subsequent processes of solution and storage as fixed into insoluble forms, or as anions on the anion exchange complex of the soil, and the subsequent degradation or leaching of the arsenic compounds from the soil.

Persistence in Soil

Depending upon solubility of the added arsenic compounds, they may remain as added(while gradually going into solution) for varying periods of time. Apparently under some conditions despite the low solubility constants of the materials this solution rate can be fairly rapid. Epps and Sturgis found that nearly one quarter of the Arsenic Trioxide they added to a silt loam soil went into solution readily. This may be more rapid where the soil is more acid, or where the soil is less aerated.

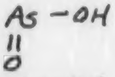
A major aspect of the persistence of the arsenic in the soil will be in the various chemical processes which occur in the soil after the arsenic is in soluble form. The arsenic in soluble form from the PAX 400 will be mainly as arsenate:



$$OH-\overset{\overset{OH}{|}}{\underset{\underset{O}{\|}}{As}}-OH$$

or H_3AsO_4

This dissociates as a very weak acid in a stepwise fashion in final dissociation with increasing acidity of the soil. In a poorly drained soil with reducing conditions this may be converted to Arsenite:



$$\underset{\underset{O}{\|}}{As}-OH$$

In soils that were kept wet for extended periods, Epps and Sturgis found that the soluble As content went up to 4.0 ppm by the third week. This was on a silt loam. This would be at a toxic level to most plants of greater than 2.0 ppm water soluble arsenic.

Anion Exchange Storage of Arsenic

Anion exchange adsorption of the soluble arsenate may take place in soils which have a high anion exchange capacity. This would represent a type of storage which would lend persistence to the arsenic in soil.

In the case of the prescribed addition of PAX 400 to the soil, the 21.49 grams of arsenic added per square meter represents nearly a~~n~~ gram equivalent weight of arsenic in anion form if it were all soluble. Considering a soil with moderate anion exchange capacity, which might be 10 milliequivalents per 100 grams of fine earth, and a bulk density of 1.0, an estimate of one equivalent of anion exchange capacity per square meter to a depth of 1 cm. of soil is derived. Dratschev found that clay could adsorb up to 1% of its weight as arsenate. The amount of arsenic that would enter into anion exchange storage in the soil will depend upon the type of clay present, and its amount. Kaolinitic clays typical of mature soil will have the greatest capacity to adsorb arsenic in the anion form, and montmorillonitic clays will have least (Rubins and Dean). The persistence of the arsenic stored on the anion exchange complex will depend upon the soil pH, and on the types and concentrations of competing anions present in the soil solution. There is a displacement sequence $SO_4^= > CrO_4^= > NO_3^- > AsO_4^\equiv > PO_4^\equiv > MoO_4^= > I^- > Cl^- > F^- > OH^-$ in which equivalents of the first will displace equivalents of the following. Thus additions of $SO_4^=$, or NO_3^- as fertilizer may displace arsenate that has accumulated on the anion exchange capacity of the soil. Conceivably a concentrated addition of $PO_4^=$ could do the same. Thus the arsenic could persist for a long period on the anion exchange complex to be displaced by concentrated additions of other anions.

The anion exchange capacity of the soil, and the presence of competing anions would be variable from soil to soil, and thus quite different quantities of storage of the arsenic in this form may occur. The accidental or other changes in competing anion concentration might be another unpredictable variable.

Storage of Arsenic in the Soil in Fixed Condition

Soluble arsenic in the soil may undergo the formation of insoluble compounds with iron or calcium to become "fixed" in the soil in an analogous manner to phosphorus. Soils high in iron, high in organic matter, or high in lime usually have a high capacity to fix arsenic in insoluble form. (Cooper, et al). These soils are usually the red and yellow podzolic soils and latosols, or soils high in lime as in arid areas, or those derived form Limestone. The Hagerstown Soil Series from limestone was found to have a high capacity to fix arsenic in a non-toxic form by Gile. The arsenic compound in this fixed form has a very low solubility constant. However this may change with a change in pH, or with oxidation reduction potential (Keaton and Kardos). Thus, raising the pH of a soil containing arsenic fixed with iron may result in a release of fixed arsenic into soluble form. Similarly arsenic fixed in a soil high in lime may be insoluble at neutral pH, but released if the soil is acidified. Conceivably this could happen with the addition of a sulfur fertilizer. On the other hand, Kardos has shown that a soil high in soluble arsenic can be renovated by application of ferrous sulfate which lowers the solubility of the arsenic.

Thus, in general arsenic may be very persistent in soil in which it is likely to enter a fixed insoluble form. These would be soils high in organic matter, high in lime, or in iron. Arsenic in this fixed form could be released into soluble and possible toxic form at some later date by a change in pH and subsequent change in solubility of the fixed form. Presumable a large quantity of arsenic fixed in a soil could be released in toxic quantities by such a change. Bishop and Chisholm have recommended that the arsenic status of a soil previously treated with arsenic should be ascertained before attempting to grow arsenic sensitive crops.

Degradation Products

The soluble arsenic derived from the addition of PAX 400 may in some situations undergo transformations into much more toxic degradation products. For example, Epps and Sturgis have noted that if the soil is rendered anaerobic the arsenates may be reduced to arsenites. Arsenic in the arsenite form is much more toxic to plants. This could happen where the material had been placed on a soil that later became seasonably flooded or anaerobic. The original compounds would become more soluble, and the reduction to arsenite from arsenate take place.

A degradation sequence in anaerobic soils can proceed further to arsine by some molds. Such a sequence under reducing conditions by Anonymous in the Chem. Eng. News as follows:

Arsenite → Methylarsonic acid →
dimethylarsinic acid → dimethyl arsine.

Challenger described this sequence of reactions earlier, and showed how if temporary oxidation conditions arise the dimethyl arsine might be oxidized to CO_2 and arsenate.

Thus depending upon subsequent changes bringing about anaerobic conditions to a soil having a high content of fixed or stored arsenic, this might be transformed into compounds of much higher toxicity. This could occur under flooding, or where fill material was placed over a soil, or where construction might take place over the soil. Thus it would be wise to know where large quantities of arsenic has been added to soil as with PAX 400 to avoid such possible undesirable transformations.

Physical Concentration

The arsenical compounds added in the PAX 400 product could possibly be concentrated by physical processes occuring in the soil or in soil management practices.

Wind blowing surface soil may cause surface drifting of the soil in which arsenicals have been added and thus concentrate them in the drifting material. This has been found to occur sufficient in amount to cause a noticable increase in arsenic contents in plants grown on such drifted soils according to Jacobs et al.

Surface runoff of water and accompanying sheet erosion of surface soil may accumulate and deposit arsenic rich surface soil downslope from the application area. Conceivably such deposition could occur in anaerobic environments such as wet meadows and swamps and further accentuate the concentration by detrimental degradation products such as arsines being formed.

Soil management practices which would tend to accumulate surface soil layers in localized deposits may result in local concentrations of arsenic rich soil. For example if soil were scraped from a surface for some reason and piled in local accumulation or fill, vegetation subsequently grown there might be enriched with arsenic. Vacuum devices used to collect leaves from lawns might concentrate surface arsenic rich soils if they were dry and dusty. An example of such accumulation by a lawn mower accumulating clippings and associated surface soil has been described by Buck.

These possibilities of physical processes concentrating soils enriched with arsenic and depositing them in still more concentrated situations introduces another unknown element in the control of the distribution of the added arsenic.

Biological Concentration

Vegetation growing in arsenic treated soils may accumulate arsenic. This accumulation will be mostly in the foliar parts of the plants (Jacobs). Foraging animals might utilize the browse or herbage grown upon such soils, and if confined in some way to this forage be subject to undesirable concentrations of arsenic. Jacobs et al reported foliar contents up to 19 ppm in potatoes.

Home vegetable gardens inadvertently placed on soils which had previously been treated with arsenicals in the large amounts specified for the PAX 400 product could gather high concentrations in the foilage. The effect of such past arsenical additions in soil raising the vegetative uptake would be more noticeable on sandy soils and less on heavy textured soils as indicated by the work of Gile.

Isensee et al working with model ecosystems, aquarium tanks with food chains extending from mud to plants to aquatic animals, found that arsenicals (Cacodylic acid and dimethylarsine) were accumulated more readily by organisms low in the particular food chain established in their model. However there was a low magnification in organisms higher up this food chain. PAX 400 does not add arsenicals of the types used in this work, but some of the degradation products under anaerobic conditions could be of this type. Machlis in original work and a literature review noted that where arsenic was high in the soil or nutrient medium the arsenic content would rise up to limiting toxic amounts in the plant. These limits are different for different species. He found for example 12 ppm in Sudan grass (Sorghum vulgare var. sudanense), and 1.2 ppm, for beans (Phaseolus vulgaris var. humilis) suppressed growth. Daniel found that the level at which Arsenic was toxic in soil depended upon the phosphorus content and speculated that Arsenic toxicity to plants was a phosphorus deficiency problem.

Summary

This report has been confined to the possible problems associated with the addition of arsenious oxide and lead arsenate in the quantities prescribed for the use of PAX 400 3 year crabgrass control. The following is a summary of the main points:

1. Rates of addition of PAX 400 as prescribed result in arsenic contents in the soil of up to 2100 ppm if retained within the top centimeter of the soil (as some studies indicate) may occur. If eventual leaching and fixing of this arsenical material in the top 10 cm. of soil occurs this concentration may drop to 210 ppm. Normal arsenic background in soils ranges from 1-70 ppm.
2. The arsenic added to a square meter of soil if entirely converted to anion form would occupy the anion exchange capacity present to a depth of 1 cm of that soil.
3. Soil usually has a high capacity to retain arsenic either as fixed, that is bound in insoluble compounds; or adsorbed on the anion exchange capacity of the soil. The literature indicates that up to 8000 lbs. of arsenic trioxide could be retained in the top 9 inches of an acre of soil.
4. The arsenicals added in PAX 400 will go into solution as arsenates in well drained soils and possibly arsenite in poorly drained soil. Solution will be more rapid in acid soils and in poorly drained soils.
5. Mature soils that are heavy textured and characterized by Kaolinitic clays will adsorb large quantities of arsenate on the anion exchange complex. This may be subject to later displacement by other anions such as sulfate and nitrate.

6. Soluble arsenic in the soil may be fixed as compounds of low solubility in the presence of large amounts of iron or calcium. The solubility of arsenic in this fixed form is a function of pH. A change of pH as by liming an iron rich soil high in fixed arsenic may release large amounts of arsenic in soluble form.

7. Soils will vary widely in their capacity to store arsenic in fixed form. Light textured, low organic matter, and low iron or calcium soils will have the lowest capacities to fix arsenic in insoluble storage.

8. Degradation products of the added arsenic compounds may be particularly toxic if the soil is anaerobic (i.e. due to flooding, paving, or filling). In these situations dimethylarsine and arsine may result due to micro organism activities in the anaerobic environment. These are much more toxic than the original arsenicals.

9. Physical concentration of the arsenic rich soils by erosion and deposition may result in concentration and accumulation in new locations.

10. There may be concentration of arsenic in the foliage of plants growing on soils in which arsenic content has become locally enriched. Animals browsing or grazing this foliage may thus pick up large amounts of arsenic.

Recommendations

1. Due to the high application quantity of the PAX 400 material and the obvious high concentration of arsenic resulting in the soil from this application, and the uncertainties of later changes in the soil with possible rendering of the arsenic in available and toxic forms to plants or animals it is recommended that this product be used only with instructions for extreme care.

2. Where use is necessary, it would be best to have the application carried out under the advice and responsibility of a person knowledgeable of the possible persistence and deleterious transformations that may occur in the soil at the location.

3. A record should be kept of all applications of the PAX 400 materials so as to avoid future alterations of treated soil which may tend to concentrate or transform the arsenical compounds into more toxic amounts of forms. This will be increasingly important as multiple applications of PAX 400 are made in the same area.

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Report to PAX Arsenic Advisory Committee

May 14, 1973
A. E. Hiltbold

Weed Problems for Which PAX is Used

Vigorous, well managed turfgrass is a thing of beauty and pleasure as a home lawn, on a playing field, golf course, or in the park. Recent trends of suburban living, increased income and leisure time have enhanced the desire for attractive and useful turf. Research has expanded rapidly in selection and adaptation of grass species, nutritional and cultural requirements, and pest control. The wide array of herbicides introduced during the past twenty years has greatly improved the control of weeds in turf, yet there is not the ideal herbicide applicable to all situations.

PAX 3 Year Crabgrass Control (Reg. No. 3234-3, Dec. 14, 1970) is used primarily for control of smooth crabgrass (Digitaria ischaemum Schreb.), large crabgrass (Digitaria sanguinalis L. Scop.), and annual bluegrass (Poa annua L.) in established turfgrasses such as Kentucky bluegrass (Poa pratensis L.), red fescue (Festuca rubra L.), and bentgrass (Agrostis sp). The herbicidally active ingredient of the PAX product is the arsenic provided by As_2O_3 (25.11%) and $PbHAsO_4$ (8.25%), functioning as a preemergence material applied at the currently recommended rate of 20 lb/1000 ft^2, providing the acre equivalent of 181 and 43 lb. total As and Pb, respectively. This rate may be reduced to one-third for annual maintenance after the first year application at full rate.

Weeds generally invade where turf is thin or bare soil is exposed by heavy traffic, improper fertilization or mowing, or by poor soil moisture conditions. Crabgrass and *Poa annua* make unpleasant contrasts of color, texture, and height in the desired turf and also contribute to the thinning of the turf through competition. *Poa annua* is widely distributed in United States, appearing with germination of seed in the fall and during mild periods in the winter and spring. Most vigorous growth occurs on moist, fertile soil, but it persists in areas of low fertility and soil compaction. Seed is produced during most of the growing season; the seedheads are unsightly, difficult to mow, and contaminate adjacent areas. During the mid-to late-spring, competition from *Poa annua* retards the growth of the established turf. With onset of hot, dry weather, *Poa annua* dies rapidly, leaving a patchy, unthrifty appearance. Extensive die-back of *Poa annua* occurs even in the northern United States, making its management as a turfgrass impractical.

Crabgrass commonly begins its growth period in late spring, taking over areas left by dying *Poa annua*. Smooth crabgrass is prevalent in northeastern United States while large crabgrass is most abundant in the mid-Atlantic and southeastern states. Both species grow rapidly with abundant moisture, fertility, and light during the summer, causing severe competition of desirable turfgrass and unattractive contrasts of color and texture in the turf. Seed are produced from mid-summer until the plants are killed by frost. The dead crabgrass areas in the turf are ugly during the fall and provide the access for germinating *Poa annua* in the next cycle. Light enhances germination of both *Poa annua* and crabgrass in areas of thin turf.

Turf weeds are seldom controlled by a single procedure. Most weed control programs stress management for better turf cover and the use of herbicides. A comprehensive look at turf weed control methods would include the use of weed-free seedbeds, weed-free propagation materials, prevention of weed germination or emergence with preemergence herbicides, reducing weed growth and development with management or herbicides, regular mowing to destroy weeds intolerant of mowing, preventing seed set, and destroying the established plants (15).

Control of crabgrass requires prevention of seed set for several years. Germinating seedlings can be eliminated through competition of dense growth of the established turf and with use of preemergence herbicides.

Effectiveness of PAX 3-Year Crabgrass Control and the Availability and Effectiveness of Alternative Control Measures

PAX functions as a preemergence herbicide with considerable selectivity. The dissertation research of Stadtherr (42) is probably the most comprehensive study of PAX 3-Year Crabgrass Control available in the literature. When PAX was applied at the recommended rate of 25 lb/1000 ft^2, the product provided complete control of crabgrass the first year and residual control exceeding 90% in each of three succeeding years, with no visible injury to bluegrass. The lethal effect of PAX on crabgrass was associated with germination and emergence of the radicle. However, it was possible to seed bluegrass on a loamy soil immediately after application of PAX at the recommended (1X) rate without injury to the seeded bluegrass. On two other soils some reduction of stand occurred when seeding was done on the day of PAX application. Stadtherr found the injury by arsenicals to be due to injury of plant roots without appreciable translocation of arsenic to the above-ground parts. For example, established crabgrass did not absorb sufficient arsenic to kill the plants. Roots and stolons failed to grow into PAX-treated soil. The long period of residual activity of PAX was associated with resistance to leaching of arsenic from the soil surface. In controlled leaching experiments with PAX applied to the surface of two soils, less than 6% of the toxic substances of PAX or their equivalent arsenicals moved below the 1/2" depth after leaching with 16" of water.

Campbell and Quinlan (5) compared five preemergence herbicides including PAX for crabgrass control in Kentucky bluegrass. In comparison with sesone (2-(2,4-dichlorophenoxy) ethyl sodium sulfate), neburon (1-butyl-3-(3,4-dichlorophenyl)-1-methylurea), alanap 1-F (1% N-1 naphthyl phthalamic acid), and chlordane (octachloro-4, 7-methanotetrahydroindane), PAX at 25 lb/1000 ft^2 was the only material that gave good control of crabgrass throughout the germinating period. A continuation of the work the following year compared the PAX treatment with lead arsenate at 10 and 20 lb/1000 ft^2. Results were not outstanding, but these treatments gave considerable reductions in number of both crabgrass species.

Both pre- and postemergence herbicides were compared for crabgrass control in mixed turf in southern California (53). A single application of PAX at 25 lb/1000 ft^2 on March 1 gave complete control of crabgrass throughout the summer. Slight turf injury was noted one week after treatment but this was followed by improved color. Alanap 1-F was nearly as effective as PAX but required three applications of 18 lb/1000 ft^2 each. Other preemergence herbicides reduced crabgrass numbers but were far below the efficacy of PAX. The data showed the necessity for application ahead of crabgrass seed germination. The persistence of PAX and the short period of activity of the synthetic organic herbicides work to the advantage of PAX in this situation. Among postemergence herbicides DSMA (disodium methanearsonate) at 6.7 oz/1000 ft^2 and PMAS (10% phenyl mercuric acetate) at 2.5 oz/1000 ft^2 reduced crabgrass populations but several carefully timed applications were required.

One of the most widely recommended preemergence herbicides is DCPA (dimethyl tetrachloroterephthalate). Watschke (48) evaluated various experimental herbicides in comparison to DCPA for crabgrass control in Kentucky bluegrass and creeping red fescue turf. DCPA at 10 lb/A applied in May provided essentially complete control of crabgrass without injury to the bluegrass and only slight thinning of the fescue. Engel and Bussey (14) compared DCPA at 12 lb/A, siduron (1-(2-methylcyclohexyl)-3-phenylurea) at 12 and 18 lb/A, bensulide (O,O-diisopropyl phosphorodithioate S-ester with N-(2-mercaptoethyl) benzenesulfonamide) at 10 lb/A as preemergence herbicides applied to Kentucky bluegrass turf in late April. All herbicides attained one or more ratings of 90% crabgrass control or higher. No turfgrass injury was observed with use of these herbicides.

Daniel (10) listed the crabgrasses and Poa annua among those plants sensitive to arsenic. He points out the tolerance to arsenic of established turfgrasses such as Kentucky bluegrass, bentgrasses, and redtop. Even seedlings of these species survive arsenic applications that are toxic to crabgrass. PAX at 25 lb/1000 ft^2 (equiv. to 5.16 lb As/1000 ft^2) was compared with calcium arsenate (equiv. to 3.2 lb As/1000 ft^2) and lead arsenate (equiv. to 5 lb As/1000 ft^2) for reduction of crabgrass with similar success. Calcium and lead arsenates were effective in controlling Poa annua in turf, where these arsenicals were compared with numerous experimental herbicides in 1955-56 (21). It was concluded that for control of Poa annua in fine-leaved fescues and bentgrasses, 2.75 to 4.6 lb As/1000 ft^2, concentrated in the soil surface, offered the greatest degree of success. Freeborg and Daniel (19) have related the arsenic susceptibility of Poa annua to temperature and light conditions and to level of extractable arsenic in the soil during germination. At various levels of applied As_2O_3, toxicity was more pronounced at 30 C than at the lower temperatures. At conclusion of the growth period, 30 days after application of As_2O_3 and seeding, dilute acid-extractable arsenic was measured in the soil, using Bray P-1 extractant (0.03 N NH_4F + 0.025 N HCl). At rates of applied As_2O_3 up to 1280 ppm As in the soil, approximately 2.5% appeared in the dilute acid extract. With 30 C growing temperature, Poa annua was killed with extractable arsenic levels of 14.5 ppm As, and severely stunted and chlorotic with 8 ppm As. These results suggest that to maintain control of Poa annua at 30 C the concentration of weak acid extracts should be in the range 8-15 ppm As. At lower temperatures approximately 20-45 ppm As was required.

Translation of these levels of extractable soil arsenic to rates of application for weed control is difficult because of the locallization of applied arsenic in the soil surface and its slow rate of dissolution and movement into the soil with leaching. Water solubilities of As_2O_3 and $Ca_3(AsO_4)_2$ are 2.04 and 0.013 g per 100 ml water at 25 C, respectively. Water solutions in equilibrium with solid phases thus contain 15,450 ppm As in the case of As_2O_3 and only 49 ppm As in the case of $Ca_3(AsO_4)_2$. Arsenic concentrations in soil solution would be affected also by equilibria with soil minerals such as kaolinite and hydrous oxides of iron and aluminum. In soil immediately below the applied arsenic, where grass weeds germinate, solution concentrations must approach the saturation level.

Partial control of Poa annua can be achieved by management favoring a dense turf in late summer prior to germination of annual bluegrass. Suppression of annual bluegrass with lead or calcium arsenate has been practiced for a long time. Calcium arsenate is considered one of the most effective materials available; however, the development of a toxic level for weed control often reduces the established turf or makes reseeding unsuccessful. The activity of calcium arsenate for Poa annua control is decreased by high levels of phosphorus in the soil (31). Trial and error have been relied on in determining treatment rates.

Jagschitz (28) compared bensulide, lead arsenate, and calcium arsenate for preemergence control of Poa annua in Colonial bentgrass (Agrostis tenuis Sibth.) putting green turf. The herbicides were applied annually at the rate of 0.34, 24, and 4.8 lb/1000 ft^2 for bensulide, lead arsenate, and calcium arsenate, respectively, for a 4-year period. After several years of these treatments good control of Poa annua was obtained.

Among the more recent preemergence herbicides, benefin (N-butyl-N-ethyl-α,α,α-trifluoro-2,6-dinitro-p-toluidine), bensulide, DCPA, and nitralin (4-(methylsulfonyl)-2,6-dinitro-N,N-dipropylaniline) can prevent germination of annual bluegrass. However, they have not demonstrated broad effectiveness nor sufficient control. They seem to have little merit in cooler, humid regions where annual bluegrass may survive from year to year. While their damage to established turf may not exceed that of the arsenates, they can be expected to inflict undesirable amounts of injury on sensitive species such as bentgrass (15).

In summary, PAX is an effective herbicide for crabgrass and Poa annua control. It is more persistent than any of the synthetic organic herbicides, guaranteeing weed control for a 3-year period following a single application. Additional advantages are offered in its control of various broadleaf weeds, white grubs, and earthworms in turf. Inorganic arsenicals are currently the only herbicides providing gradual, selective control of Poa annua in closely mowed perennial turf such as on golf greens. Granular form of calcium arsenate is presently the most widely used arsenical for this purpose. Professionals in turf research, golf course superintendents, and numerous home owners have attested to the effectiveness of PAX and the arsenical approach to turf weed control. However, other herbicides are available for many situations. These materials are more demanding as to their rate and time of application and control a more limited spectrum of weeds for shorter periods of time. The PAX product offers economy in the cost of material and infrequency of application.

Behavior of Arsenic in Soil

Concern with arsenic in agricultural soils of the United States stems from crop injury associated with long-term usage of high rates of lead and calcium arsenate for insect control. Lead arsenate was used for many years in apple orchards to control codling moth. It has been estimated (2) that 20% of all the lead arsenate used in United States was used in eastern Washington. The peak use came in 1943 when applications in Washington averaged 50 pounds of arsenic per acre of bearing orchard. In 1948, DDT replaced lead arsenate for insect control. While established orchards seldom showed toxic effects of accumulated spray, replanted apple, pear, and peach would not grow in old orchards. This injury was readily apparent during the 1930's when extensive removal and replanting of orchards was done. At this time crop injury was observed in the South as a result of soil arsenic accumulations from calcium arsenate applied for boll weevil control in cotton. Dorman and Coleman (13) estimated the amount of calcium arsenate applied at from 3 to 10 pounds per acre, with as many as 6 applications per season, depending upon the size of the cotton and insect infestation. Probably not more than 30 pounds per acre were applied annually (11 lb As/A). While cotton tolerated considerable arsenic levels, injury was observed in rice (16), cowpeas and oats (8) grown on treated soil following cotton. Analyses of soil samples collected from all regions of United States and parts of Canada (4,22,29,24,37, 39,49,50) indicate that arsenic occurs in all soils regardless of pesticide application. Amounts found in soils receiving no arsenical treatment range up to 14 or 18 ppm As (49,4,39) or may average as much as 13 ppm As (50). A recent survey of arsenic contents in more than 500 soil samples from Alabama (24) had a frequency distribution of soils with 14% containing less than 1 ppm As, 46% with 1-4 ppm As, 22% with 4-7 ppm As, 14% with 7-10 ppm As, and 4% with more than 10 ppm As. These samples were selected from among those submitted for soil test and fertilizer recommendations for cotton, with the objective of determining the current arsenic levels in fields presumably treated with calcium arsenate in the past. The data indicate that these fields have not received appreciable calcium arsenate or else that extensive loss of arsenic has occurred through erosion, crop removal, or leaching below the plow layer.

The more extreme concentrations of arsenic occur in orchard soils where levels in excess of 100 ppm As have been reported (4,22,37,49). Woolson et al (50) found arsenic residues averaging 165 ppm As in samples of 58 surface soils with history of arsenic application. Washington orchard soils (10 samples) averaged 627 ppm As, with one as high as 2,500 ppm As. Significantly greater amounts of arsenic occur in soil beneath the trees, compared to soil between the trees, as a result of spray drip (37,50). Analysis of four commercial orchard sites in Oregon in 1945 showed as much as 116 ppm As and 377 ppm Pb in treated soil, amounting to 20 to 30-fold increases in arsenic and 13 to 40-fold increases in lead as a result of 20-25 years of lead arsenate spraying (30).

Where records of past usage of arsenicals are available, recent determinations of arsenic residues in soil indicate the element to be highly persistent in soil. The level of arsenic established in soil (126 ppm As) with annual applications of lead arsenate remained essentially constant during 5 succeeding years without further addition (34). Arsenic levels injurious to red pine in forest nursery soils were estimated to be little changed from those established 35 years previously when As_2O_3 was used for white grub control (44). The recovery of applied arsenic in soil of Indiana orchards was nearly complete (50), indicating very little arsenic leached or lost. On the other hand, arsenic concentrations established with lead arsenate in 1935 for white grub control in turf (38) underwent continuous decline during the following 15-year period. The rate of loss was related directly to arsenic concentration in the soil. The gradual decline of grub control during the period was associated with a corresponding decline in soil arsenic. Leaching of arsenic has been found to be a significant loss process in soils with low contents of active iron and aluminum. Losses of applied arsenic from sandy surface soils in the Netherlands were directly related to the amount of arsenic in the soil, with an average half-life of 6.5 years (45). Much of the arsenic lost from the upper 20 cm of soil was found in the 20-40 cm depth, although net loss of applied arsenic occurred continuously. Leaching was concluded to be an important factor in dissipation of toxicity from high rates of sodium arsenite applied to a loamy sand soil in New Jersey (18). Complete inactivation of the 75 lb As per acre application was observed in the upper 4 inches of soil during the 30-month sampling period. At the other extreme, McGeorge (35) observed the complete retention of arsenic within the upper 4 inches of ferruginous Hawaiian soils sprayed with sodium arsenite equivalent to 3.75 lb As per acre for a 5-year period, despite rainfall averaging 200 inches per year on flat, porous soil surfaces. Arsenic applied as methanearsonates over a 4-year period for weed control in turf on a sandy loam soil in Alabama was recovered in large part from the upper 6 inches of soil, but significant leaching into the 6-12 inch depth was observed (29). Soils differ widely in those properties affecting arsenic retention or leaching, and those properties similarly affect the capacity of soil to inactivate toxic applications of arsenic.

Crafts and Rosenfels (9) found that red soils had greater capacities for reducing arsenic toxicity than soils of other colors in California. Similarly, Cooper et al (8) in South Carolina showed that coarse-textured gray soils such as the Norfolk and Durhum series were seriously affected by relatively light applications of calcium arsenate, whereas fine-textured dark soils such as Cecil, Greenville, and Davidson series were not affected, even at high application rates. Dorman and Coleman (13) applied rates of calcium arsenate up to 1600 lb per acre prior to planting cotton on soils varying in texture from sandy loam to clay. Only on Ruston sandy loam were cotton yields depressed by arsenic during 4 years of cropping after application. The fourth year yields showed the 400 lb rate of calcium arsenate was completely inactivated and much of the toxicity of the 1600 lb rate was lost. Gile (20) compared the effects of colloidal material from 36 soils and subsoils on the toxicity of calcium arsenate in greenhouse pot experiments. He concluded that the resistance of a soil to calcium arsenate injury depends on the quantity of colloid present and the specific resistance of the colloid, the latter being dependent upon the quantity and reactivity of the iron present. Recent attempts to characterize the chemical forms of arsenic in soils (29,50,51) indicate that most of the residual arsenic is bound as insoluble iron and aluminum compounds.

Over the range of soil pH from 4-8 the predominant arsenate ion is $H_2AsO_4^-$ with $HAsO_4^=$ increasing with pH above 7. With mildly reducing conditions in the pH 4-8 range arsenious acid (H_3AsO_3) becomes stable (17). Addition of iron oxide to an arsenate-arsenite system results in oxidation of arsenite to arsenate with a subsequent increase in the redox potential (32). These chemical factors in aerobic soils favor the transformation of reduced forms of arsenic to the predominant arsenate form. Most soils contain sufficient reactive iron and aluminum to precipitate arsenate in very insoluble forms (51). The time required for added arsenate to equilibrate with these insoluble forms varies with rate of application and the soil content of reactive iron and aluminum, but generally is marked by rapid decline of water soluble arsenic and more gradual shift of arsenic into the iron fractions. When arsenic is displaced from field-weathered, high arsenic soil by leaching with phosphate, the more soluble aluminum arsenates are removed, concentrating the less soluble iron arsenates (51).

Microbiological transformations of arsenic are known to occur. Quastel and Scholefield (40) reported the microbiological oxidation of arsenite to arsenate in soil. The course of arsenite oxidation was found to follow the typical log growth curve, with oxygen uptake in agreement with the reaction $NaAsO_2 + H_2O + O \rightarrow NaH_2AsO_4$. Addition of 0.1% sodium azide completely inhibited arsenite oxidation. Upon enrichment of the microbial population with arsenite-oxidizing species, subsequent applications of arsenite were oxidized rapidly and without the initial lag phase. Green (23) isolated bacteria, *B. arsenoxydans* and *B. arsenreducens*, from solutions of arsenite in cattle dipping tanks. These fecal bacteria were found capable of oxidizing and reducing arsenic and were enriched in the 0.1% sodium arsenite of the dipping tanks. Microbiological reduction of arsenate with evolution of arsenical gases was reported by Thom and Raper (46). In addition to numerous strains of *Aspergillus* and *Scopulariopsis* found to evolve the odor of arsine, other active fungi were isolated from arsenic-toxic field soils. In view of this capability of soil fungi it was concluded that accumulation of arsenic in soil may be expected to occur only under massive amounts or under special conditions unfavorable for development of a varied microflora. Later studies by Challenger and co-workers (3, 6) verified the conclusion of rather widespread capability for arsine evolution among fungi and also identified the gaseous products. Fungi were grown on bread cultures containing 0.1% of As_2O_3, Na_2HAsO_4, $CH_3AsO(ONa)_2$ disodium methanearsonate, $(CH_3)_2AsO(ONa)$ sodium cacodylate, $C_3H_7AsO(ONa)_2$ sodium propylarsonate, and others. Cultures were aerated and the evolved gases absorbed and determined. Strains of *A. niger*, *A. glaucus*, *S. brevicaulis*, *Penicillium notatum* and *P. chrysogenum* were active on one or more of these arsenicals. Trimethylarsine ($(CH_3)_3As$) was the most common gaseous product, accounting for loss of 70% of the As_2O_3 during a 24-month period. In all cases the arsenic was reduced to the +3 oxidation state and all substituent oxygen atoms replaced by methyl groups. With the methanearsonates and propylarsonates the evolved arsine contained the original alkyl intact: trimethyl arsine and dimethyl-n-propylarsine. Zussman et al (54) reported the production of a volatile arsine by the dermatophyte Trichophyton rubrum when grown in arsenate-containing media. Arsenite did not serve as a substrate for arsine production. While only aerobic microorganisms have thus far been identified in production of methyl arsines, losses of arsenic by volatilization from anaerobic soils have been reported (16, 41, 52). Woolson and Kearney (52) found extensive volatilization of arsenic from aerobic soils treated with cacodylic acid, and even greater losses from anaerobic soils. The volatile product was considered dimethyl arsine, unstable and readily oxidized in air to the oxide or to cacodylic acid. The ultimate fate of the arsenic was concluded to be metabolism to arsenate and inactivation by soil iron compounds. Oxidation of the methyl substituents of arsenic has been reported to occur slowly in soil (12,47,52) with liberation of CO_2 and arsenate.

Reduction of ferric iron associated with bacterial growth in submerged soils may be implicated in the observed sensitivity of rice to arsenic residues in soil from use of calcium arsenate in cotton. While no injury of cotton occurred with use of the arsenical, frequently toxicity appeared after the soil was flooded during growth of rice (41). Reduction of iron arsenate at the low prevailing redox potential would be expected to increase the concentration of soluble arsenate and arsenite. Similarly, the toxicity of As_2O_3 used for weed control in forests of the eastern United States was found to be greater on wet, poorly drained soils than on well drained sites (33).

Plants generally absorb and translocate relatively small amounts of arsenic into above-ground parts. McLean et al (36) investigated the potential hazard of vegetable gardening on soils formerly in lawns treated with lead arsenate for white grub control. Field plots were established on Sassafrass loam in New Jersey, with rates up to 1000 lb of lead arsenate per acre incorporated in the upper 4 inches of soil 7 days before planting a variety of vegetables. Arsenic analyses of the harvested produce showed less than 1 ppm As in all plants. On a former orchard site contaminated from arsenic sprays 10 years previously, the greatest concentration of arsenic observed in vegetables was less than 2 ppm As in onion tops, despite the soil arsenic concentration of 233 ppm. Oregon orchard soils with arsenic contents elevated 20 to 30 times original level by accumulated spray residue produced a variety of forage and vegetable crops with arsenic contents increased only 1.3 to 3.0 times over those from adjacent low arsenic soils (30). Root portions of the plants were more affected by soil arsenic than were tops, being increased 4-15 times over control level. However, the edible parts of plants grown in sprayed-over soils contained on the average less than a tenth of the legal tolerance for arsenic on fruits and vegetables. Williams and Whetstone (49) determined arsenic contents of numerous plant species growing on soils with accumulated arsenic and others without arsenic histories. In untreated soils various vegetable and weed species contained less than 2 ppm As. In a Sassafrass sandy loam with 270 ppm As in the upper 3 inches, orchard grass and wild carrot contained 2 ppm As or less. Mustard was the only species containing appreciable arsenic· 34 ppm in roots and less than 1 ppm As in tops. The arsenic contents of lawn grasses ranged up to 2 ppm As on soils treated repeatedly with As_2O_3 and lead arsenate to establish levels from 150-550 ppm As in the soil over several years. Johnson and Hiltbold (29) reported arsenic contents of field crops grown on a sandy loam soil containing residual arsenic from methanearsonate applications in turf. With arsenic levels in the surface soil ranging up to 28 ppm As no effects on crop yields were obtained, but arsenic residues were found in harvested material. Arsenic uptake was related to soil arsenic concentration and differed among crops. Seed of cotton and soybeans accumulated the highest concentrations of arsenic, up to 9 ppm As. Vegetative material of sorghum was in an intermediate group, and the winter forage legumes and grasses were in the lowest arsenic group containing 2 ppm As or less. The low As content of corn grain provided an interesting contrast with the oilseed crops. Differences in arsenic uptake by warm- vs. cool-season crops was postulated to be related to their rates of transpiration. A similar effect of season was observed by Benson (1) with barley growing in greenhouse pot experiments with arsenic-contaminated soils. Sand cultures with high rates of lead arsenate severely curtailed the growth of barley in summer but had little or no effect in winter. Jacobs et al (27) found the order of crop tolerance to arsenic to be potatoes > peas > sweet corn > snap beans grown on Plainfield sand in Wisconsin after rates up to 643 lb As/A were applied as sodium arsenite. While yield reduction of the latter 3 crops occurred at arsenic concentrations of as low as 10 ppm As in this soil, arsenic concentrations in the edible portion of these crops was less than 1 ppm As. Flesh of potato tubers contained up to 0.5 ppm As and only the peelings showed significant arsenic contamination (up to 84 ppm As). A subsequent survey (43) of 18 Wisconsin potato fields known to have been treated previously with sodium arsenite as a vine killer in potatoes showed that while arsenic levels in soil ranged from 2.2 to 25.7 ppm As, the arsenic content of potato tuber peelings was only 0.2-2.6 ppm As and up to 0.6 ppm As in tuber flesh. Deuel and Swoboda (11) studied the yield and arsenic content of cotton and soybeans as indicators of the toxicity of As_2O_3 applied to 2 soils. While 50 lb As/A was sufficient to decrease the vegetative growth of cotton in Amarillo fine sandy loam, as much as 500 lb As/A was required in the Houston Black clay. An internal arsenic concentration of only 4.4 ppm As in the plant tissue caused a growth reduction of cotton on both soils. Only 1 ppm As in the tissue of soybean plants induced a growth reduction.

The similarity of phosphorus and arsenic, and the possibility of applying phosphorus to ameliorate toxicity of arsenic in soils has attracted considerable attention. Hurd-Karrer (25) conducted nutrient culture experiments to determine the effect of phosphate on toxicity of arsenic to wheat. She suggested that arsenates would be non-toxic if the P:As ratio were more than 4:1. Results of Clements and Munson (7) supported the concept of antagonism of phosphate on arsenate absorption. Phosphorus in culture solution was found to effectively inhibit absorption of pentavalent arsenic but not in reducing the toxicity of the element within the plant. Jacobs and Keeney (26), however, found that increasing rates of phosphorus aggravated arsenic toxicity in Plainfield sand and resulted in increased arsenic uptake by corn. Tissue concentration of arsenic was about 10 times higher in corn grown on Plainfield sand than on Waupun silt loam with equal levels of applied arsenic. Similar results were reported by Woolson et al (51) for the Lakeland sand and Hagerstown silt loam. Application of phosphorus equal to arsenic in Lakeland sand increased the toxicity to corn over that from arsenic alone. In Hagerstown silt loam phosphate reduced arsenic toxicity. When available P and As were determined by extraction of the soil it was found that better plant growth was associated with P:As ratio of 6.8 than with P:As = 0.7 or 3.3, supporting the conclusions of Hurd-Karrer. However, increasing phosphate resulted in increased arsenic contents of the plants despite their improved growth.

In summary, arsenic is an ubiquitous constituent of plants, ranging from trace levels to perhaps 40 ppm As in certain severely injured plants. Arsenic is not extensively translocated from roots to tops and the nature of root injury to plants seems to preclude large uptake of arsenic. Furthermore, the threshold for arsenic injury in above-ground parts of plants is low; thus restricted growth limits arsenic accumulation in harvested portions. In order for most soils to sustain available arsenic concentrations sufficient to induce injury, extreme contents of total arsenic are usually required. Because of extensive adsorption and precipitation in insoluble form, arsenic availability decreases rapidly in soil. Leaching functions in some very sandy soils to slowly move arsenic into deeper, more clayey subsoil. Microbiological transformations occur in both aerobic and anaerobic soils to produce methyl arsines that probably account for appreciable losses of applied arsenic from soils. There is no evidence of biomagnification of arsenic in the environment.

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THE THREE-YEAR PAX CRABGRASS CONTROL PRODUCT: EFFECTS ON FISH AND WILDLIFE; BIOCONCENTRATION

Eugene H. Dustman

Introduction

In duscussing the role which the Three-Year Pax Crabgrass Control product may play in producing an effect on animals, it is fitting to proceed from the general to the specific. Much of the information basic to an understanding of the material in this section, or, indeed, any portion of the Committee's report pertaining to environmental considerations, must come from various sections of the report dealing with the chemical and physical characteristics of arsenic, its toxicological properties, and its effects on and behavior in soils, plants, and animals.

Throughout the course of this assignment there has been the constant urge to extend one's self beyond the limits of the Committee assignment, and to address the much larger problem of arsenic in the environment. This, of course, transcends the responsibility of the Committee; therefore, a true and accurate assessment of conditions resulting from application of the Pax material cannot extend much beyond the limits of the area to which it is applied unless other, and surely more important, sources of arsenic and lead are considered.

Arsenic is a ubiquitous substance in our environment. It occurs in many chemical combinations, some organic, some inorganic. It also occurs in elemental form. Its chemical combinations under natural conditions remain largely unidentified and poorly understood because the current most sensitive analytical methodology does not make their separation possible. In studies of the significance of arsenic in animals, plants, and the biosphere generally, the literature is replete with measurements of total arsenic; yet it is believed that the levels at which arsenicals are bound in animal tissues is proportional to their toxicity (25), and toxicity generally is related to the valence of the arsenic (31).

Pentavalent arsenic is generally less toxic than the trivalent form (48); organic forms are generally much less hazardous than the inorganic forms (30). Frost (19) points out that factors such as the degree of absorption into the cells, the rate of metabolism, and the rate of excretion vary with circumstances and cause variations in toxicity. He uses a striking example to support the view that the levels at which arsenicals are bound in tissues is proportional to their toxicity: At their respective LD_{50} levels of 700, 16, and 0.08 mg/kg tryparsamide, benzenearsonic acid, and benzenearsenoxide produced similar levels of arsenic in rabbit tissues. The same held for other animals. He concluded that target enzymes in the rabbit, although far more susceptible to trivalent (benzenearsonic acid and benzenearsenoxide) than to pentavalent (tryparsamide) arsenicals, bind a similar level of arsenic at point of death. These differences clearly relate to the rate at which the arsenicals are excreted.

Good general orientation to the subject of arsenic is to be had by referring to publications by Frost (19) and Schroeder and Balassa (48). Arsenic is found in soils, most foods, many waters, almost all plants and plant products, and in most animal tissues. Two forms occur in man's environment, pentavalent and trivalent. Biological activities in these valence states differ markedly. Pentavalent arsenic as arsenate (inorganic arsenic acid or its salts) is nontoxic in normal concentrations, is excreted rapidly, largely through the kidneys, probably does not accumulate to any great extent in human tissues, is a normal constituent of food and may (possibly) perform some physiological function. Schroeder and Balassa refer to it as biogenic arsenic. Most ensymes are not inhibited by it, and it can substitute for phosphate in some phosphorylases. Human daily intake is in the range of 400 - 1000 micrograms. However, as Frost (19) points out, "Although As^{+3} compounds are more toxic than their As^{+5} analogues, after autoxidation an excess of either would be expected to intefere with phosphorous metabolism. Such a mechanism may account for the additive toxicity of different arsenicals."

Trivalent arsenic as arsenite (inorganic arsenous acid and its salts) is the principal form produced commercially. It is toxic; it chelates with dithiol groups and inhibits those ensymes dependent thereon. It accumulates in animals, is a contaminant of soils and foods through its use in herbicides and other pesticides and performs no known physiological function. Oxidation of trivalent arsenic to the pentavalent form occurs slowly in the upper layers of the earth's crust and in animals.

Various workers have either suggested or concluded that pentavalent arsenicals are reduced in vive to the toxic trivalent form (22, 27). Until such time as this matter is resolved, there remains the fundamental question in arsenic metabolism of whether arsenicals accumulated in body tissues can be stored as relatively innocous residues. And of much interest is the recent work of McBride and Wolfe referred to in Chemical and Engineering News (9). These workers have found that through the action of anaerobic methanobacteria arsenic is converted into the highly poisonous dimethyl arsine, which may behave in aquatic systems in much the same manner as methylmercury. With methyl cobalamin serving as the methyl donor, $As^{+5} \xrightarrow{\text{(reduction)}} As^{+3}$ (arsenite) $\xrightarrow{\text{(methylation)}}$ methyl arsonic acid $\xrightarrow{\text{(reduction)}}$ dimethyl arsinic acid (As^{+}) $\xrightarrow{\text{(reduction)}}$ dimethyl arsine (As^{-3}).

It has long been known that natural arsenate may be reduced to toxic compounds (AsH^3) by heat or by the action of certain algae and molds (19, 48).

Toxicological Considerations

Although many different species of animals are exposed to arsenic when the arsenicals are released into the environment, detailed toxicological investigations have been confined largely to man, a variety of his domestic animals, and common laboratory forms which lend themselves well to detailed experimentation and observation. The findings made through studies of these forms relate generally to many other forms. Oehme (38) and Lisella et al. (32) offer abbreviated accounts of the mechanisms of inorganic arsenic toxicity. Oehme's discussion occurs under the headings "absorption", "distribution", biotransformation", and "excretion".

Absorption: Certain forms of arsenic may be absorbed through the intact skin. Necrosis and ulcerations may appear at the site of contact; and cell division may be inhibited and nuclear abnormalities occur. Absorption from the digestive tract is dependent upon solubility. Finely divided powders are more completely absorbed than coarse ones. Gastric juices may enhance solubility, especially the carnivores with their highly acid digestive tracts. Soluble compounds of arsenic are well absorbed through the digestive tract and all mucous surfaces including the lungs (by inhalation). Some victims may exhibit gastrointestinal disturbance; others may show nervous manisfestations.

Distribution in body: After absorption, 90 - 95% of the inorganic arsenic is found in red blood cells in combination with hemoglobin; that in serum is bound to proteins. Within 24 hours it rapidly leaves the blood (the rat is a notable exception) and goes to the liver, kidney, lung, wall of the gastrointestinal tract, and spleen. Smaller amounts may be found in muscle and nervous tissue. In about 2 weeks, or with continuous exposure, arsenic will begin to build up in skin, hair, and bones. Chronically poisoned dogs have four to six times more arsenic in hair than in liver per unit of weight. Arsenic binds tightly to organic sulfhydryl groups of the protein fraction of the respective tissues (enzyme inhibition) and is only slowly released. Inorganic arsenic passes across the placenta as demonstrated in the human fetus (16).

Biotransformation: Widespread damage is caused by arsenic combining with the sulfhydryl groups of proteins. Inhibition of these enzymes causes a breakdown in cellular metabolism. Serving as a substitute for phosphorous, arsenic also uncouples oxidative phosphorylation in the liver mitochrondia, thus discouraging the production of energy. Other enzyme systems also are affected. Though relatively stable, the thio-arsenic compounds may be oxidised to arsenoxides and then to other oxidation compounds.

Excretion: Inorganic arsenic is excreted mostly by the liver and the kidneys. Most of the arsenic excreted in the urine from a single dose will appear within the first 4 days. In man, arsenic excretion begins 2 - 8 hours after exposure, and it may still be found in small amounts after 10 days. Up to 70 days may be required for complete elimination after repeated administration of arsenic. A smaller amount of arsenic may be eliminated in feces.

Acute Toxicity of Lead Arsenate, Arsenic Trioxide, and Certain Other Arsenicals

Lead arsenate ($PbHAsO_4$) and arsenic trioxide (As_2O_3) (also known as arsenic oxide and arsenous oxide) are the principal ingredients in the Pax product. An examination of toxicity measurements for these compounds is in order. Those measurements associated with certain other arsenicals are included for comparative purposes.

The LD_{50} method for measuring toxicity had not been developed when inorganic arsenicals were having their heyday as insecticides in the early 1900's; thus the older toxicity measurements are not strictly comparable with modern-day LD_{50} readings. Nonetheless, these earlier measurements are useful and enlightening and in some instances the only ones available. The following material summarizes some of the more meaningful acute toxicity measurements as I was able to unearth them in the literature. The information on domestic and laboratory forms is given to provide some indication of what might be expected to affect wild animals even though distantly related.

Arsenic Trioxide

Mammals

The LD_{50} for the rat was 385 mg/kg (283 mg of As) when given an encapsulated dose of arsenic trioxide in a 4-day test (17). The LD_{50} for the rat was 150 mg/kg of As when the arsenic trioxide was fed dry mixed with protein or carbohydrate in a single oral dose over a 2-week period; it was 230 mg/kg of As when arsenic trioxide was fed in a fat diet under the same conditions (43).

The lethal dose for the cow was 15 - 45 g when arsenic trioxide was administered in a single oral dose (10). The lethal dose of arsenic trioxide was 19.4 g for horses and cattle when given a single oral dose (46).

The estimated lethal dose of arsenic trioxide for sheep was 1.04 g when given as a single oral dose to animals weighing 80 - 100 pounds (49).

Using the guinea pig as a test species, O'Kane et al. (39) found that arsenic trioxide will cause lethal poisoning in about ½ to 2/3 the dosage of arsenic administered in the form of lead arsenate. They also determined that the solvency of arsenic trioxide and lead arsenate was enhanced by the presence of gastric juices which they considered to be hydrochloric acid.

Frost (19) has stated that arsenic trioxide poisons surreptitiously, the victim consuming poisoned food with fair appetite until the end. This feature made As_2O_3 a useful rodenticide.

Birds

The lethal dose of arsenic trioxide for chickens was 5 g when given to 3 - 4 pound hens as an encapsulated single oral dose (21).

The lethal dose of arsenic trioxide for chickens is given as 1 - 3 g when it was mixed with bran as a grasshopper bait (59); only two of twenty-four died, others went off feed.

The minimum lethal dose of arsenic trioxide for chickens was 75 mg/kg when it was mixed in fine powdered form with a food mixture (57).

Fishes

The tolerance level of pink salmon fry exposed for 3 days to arsenic trioxide in aerated sea water closely approximated 9.3 ppm (26). Exposure for 10 days in aerated sea water to arsenic trioxide followed by transfer to pure running sea water indicated a tolerance level between 2.6 and 5.3 ppm for both periods. Even after initial concentrations (method did not employ a continuous flow system in which concentration would be held constant) of 5.3 and 9.3 ppm had remained in aquaria for 22 days, statistically significant kills resulted in less than 3 days, indicating that danger of arsenic trioxide poisoning can persist for some time even though the arsenic trioxide had apparently changed to a form not detectable by the analytical method used.

Lead Arsenate

Mammals

The minimum lethal oral dose of lead arsenate given in pelleted form to domestic rabbits was 200 mg/kg (40 mg of As) (8). The same dose also killed wild hares but death was longer delayed.

The lethal dose for sheep was 3.9 g when lead arsenate was given as a single oral dose. Death occurred on the 12th day (4).

Three pairs of sheep weighing 80 - 100 pounds each were fed 1, 0.5, and 0.25 g of lead arsenate per day. The two sheep on the 1 g dosage died within 7 days; one of the sheep receiving 0.5 g/day died after 35 days; one of the animals receiving 0.25 g died at the end of 35 days (35).

Birds

The lethal dose of lead arsenate for the chicken was 0.1 - 56 g when given in a single oral encapsulated dose (54). Death occurred over a span of 2 - 27 days.

Sodium Arsenite

A value judgment such as the one to be made on the Pax product should have the benefit of readings on the sensitivity of aquatic organisms to the principal components of the material. Other than for acute toxicity measurements on pink salmon (26) I have been unable to find such determinations. Rather than leave such a serious void, I am including information on acute toxicity of sodium arsenite which has been assembled largely by Pimentel (45). I hasten to add, however, that sodium arsenite, a compound commonly used for aquatic weed control, is more soluble than either of the Pax arsenicals and is roughly 9 to 11 times more toxic to mammals and two times more toxic to birds than arsenic trioxide. One must take this into account as he reads the material given below. Sources of Pimentel's information are given.

Mammals

The LD$_{50}$ for the rat was 10 - 50 mg/kg(28); and 42 mg/kg (24 mg of As) as determined by Done and Peart (17); and for the mouse, 51 mg/kg (36) when sodium arsenite was given in single oral encapsulated doses.

The lethal dose of sodium arsenite for the white-tailed deer was 4 cc of arsenic solution when given as a single oral dose(4).

The lethal dose of sodium arsenite for the cow was 1 - 4 g and for the dog 0.05 - 0.15 g when given as a single oral dose (10).

Birds

Mallard ducks tolerated 8 mg/day of sodium arsenite for a period in which the total dose reached 973 mg/kg in the birds (55).

The minimum lethal dose of sodium arsenite for the chicken was 37.5 mg/kg when mixed in fine powdered form with a food mixture and given in pelleted form (57).

Fishes

LC_{50} for various fishes

Species	Exposure Time (hours)	LC_{50} (ppm)	Source
Lake emerald shiner	24	13.5	Swabey and Schenk (52)
Spottail minnow	24	45	Boschetti and McLoughlin (3)
Bluegill	24	58	Cope (12)
Rainbow trout	24	100	Cope (12)
Rainbow trout	48	36.5	FWPCA (20)

Bond (2) reported sodium arsenite to be safe (for fish) at dosages of 2 - 4 ppm in soft waters and 5 - 6 ppm in hard waters.

Rainbow trout (LD_{50} = 60 mg/kg) and bluegills (LD_{50} = 44 mg/kg) were relatively tolerant of sodium arsenite compared with other herbicides (15).

Cope (15) reported that a dosage of 4 ppm of sodium arsenite caused kidney and liver damage in bluegills.

Sodium arsenite applied at 5 ppm for the eradication of submergent aquatic vegetation in ponds had no effect on rainbow trout or brook trout populations (29).

Molluscs and Arthropods

The minimum lethal dosages (ppm) producing a kill of fish-food organisms exceeding 25% are the following: *Daphnia*, 3.0; *Eucypris*, 6.0; *Hyallella*, 2.5; *Culex*, *Aedes*, and *Anopheles*, 6.0; and *Chironomus*, 10.0(60).

The 24-hour LC_{50} for stonefly nymphs (*Pteronarcys californica*) was 140 ppm (47).

The 48-hour EC_{50} (immobilisation value at 60 degrees F) for water-fleas (*Simocephalus serrulatus*)and *Daphnia pulex* was 1,400 ppb and 1,800 ppb, respectively (47).

Johnson (29) reported that 3 to 8 ppm of sodium arsenite killed filamentous algae and submerged aquatic plants in ponds, but had no effect on the numbers of pond invertebrates such as chironomid larvae, beetle larvae and adults (Haliplidae),true bugs (Nodonectidae and Dystiscidae), mayfly nymphs, damselfly nymphs, dragonfly nymphs, and amphipods. Walker (58), however, reported that treating ponds with sodium arsenite at dosages from 2.5 to 20 ppm caused a 50% reduction of midges, waterbugs, and snails.

The 24-hour LC_{50} for stonefly nymphs (*Pteronarcys*) was 160 ppm (11).

Phytoplankton and Zooplankton

Sodium arsenite at 4 ppm did not affect the number of phytoplankton, but did cause drastic reductions in zooplankton (14).

Lethal Residue Levels

Residue levels in tissues and organs which are indicative of lethality are a boon to the one who is attempting to determine the cause of death of an animal. Unfortunately, these levels usually do not rest within narrow limits. As a general rule of thumb, 10 to 15 ppm on a wet weight basis in the liver and kidney is indicative of arsenic poisoning if clinical signs, necropsy findings, and history are compatible (5). However, as Hatch and Funnell (24) discovered in their studies of cattle which had died of arsenic poisoning, arsenic levels in the liver and kidney were as low as 1.5 to 5 ppm and as high as 30 ppm, wet weight.

Cacodylic Acid

It has already been mentioned that inorganic arsenicals are generally more toxic than organic arsenicals. A number of the organic compounds are used as herbicides. They usually cause much less of an insult to the environment because they are less toxic and less persistent than the inorganic compounds. Toxicity data on cacodylic acid, an organic arsenical, is presented here for comparative purposes.

Mammals

The LD_{50} for the rat was 1,280 to 1,400 mg/kg to cacodylic acid when this dosage was fed orally (28).

Fishes

USDI (55) reported no effect of cacodylic acid on longnose killifish at 40 ppm in a 48-hour exposure test.

The LD_{50} for mosquito fish and taillight shiners approached 1,000 ppm for cacodylic acid (41). Largemouth bass, fed for 2 weeks on mosquito fish exposed to 1,000 cacodylic acid for 24 hours, appeared to be unaffected by the treatment.

Mosquito fish, largemouth bass, and taillight shiners exposed to concentrations of 100 ppm of cacodylic acid for 72 hours survived well.

Some mortality was observed when concentrations reached 631 ppm with an exposure time of 72 hours (41).

Amphibians

The 48-hour LC_{50} for *Bufo* tadpoles was between 100 and 1,000 ppm cacodylic acid (41).

Molluscs

Exposure of the eastern oyster to 40 ppm of cacodylic acid for 48 hours had no noticeable effect (56).

Arthropods

Cacodylic acid at 40 ppm had no effect on pink shrimp during a 48-hour exposure(56).

Two species of dragonfly nymphs (*Pantala* sp. and *Gynacantha nervosa*), exposed for up to 72 hours to 1,000 ppm of cacodylic acid, showed no noticeable effects (41).

Chansler and Pierce (7) reported that cacodylic acid injected at the rate of 1 to 2 ml per injection at 2-inch intervals around the trunk killed bark beetles (*Dendroctonus* spp.).

Pax Product

The LD_{50} for the rat was 625 mg/kg (129 mg/kg of As) of the Pax crabgrass control material when it was given as a single oral encapsulated dose (17). This study indicated that the Pax product is more than twice as toxic as arsenic trioxide when the comparison is made on the basis of the mg/kg of elemental arsenic.

Horses and cows died after eating grass clippings that were taken from lawns treated with Pax 3-year Crabgrass Control (6). Using the Pax preparation containing arsenic trioxide at 27 percent and lead arsenate at 8 percent plus 0.2 percent heptachlor Buck (6) determined that grass clippings taken from treated plots contained arsenic levels that were hazardous to mammals following a period of several months, after several inches of rain and snowfall, and after the lawn had been mowed seven times.

Sublethal Studies

Since arsenicals are used rather freely in animal nutrition as food additives, a number of sublethal studies have been made to make certain that chronic effects do not attend such use. Other studies have been made to determine sites of deposition, rates of excretion, species differences, etc. A few of these studies will be mentioned here.

Peoples (44) added a solution of As_2O_3 to the diet of three cows to give levels in the food of 0.0, 0.8, 4.0, and 20 ppm corresponding to doses of 0.04, 0.20, and 1 mg/kg. During the 8-week feeding period milk, urine, and feces were analyzed. The animals were sacrificed and tissues analyzed for arsenic and examined for pathology. No evidence of gross or microscopic pathological changes were found in the tissues. No arsenic was found in the milk, muscle, brain, or fat. Other tissues at the highest dose in ppm were liver, 2.10; kidney, 0.90; spleen, 0.60; skin, 0.25; intestine, 0.30; and bone, 0.16. Most of the arsenic was excreted in the urine; about 10% was excreted in the feces.

Groves et al. (23) fed equivalent amounts of arsenic in the form of lead arsenate (103.4 g) and arsenic trioxide (29.4 g) to pigs for 283 days. Pigs were sacraficed at the end of the experiment and autopsies and tissue analyses were performed. No pathological changes were noted; the blood picture appeared normal; and pig weights did not differ significantly. The pig fed lead arsenate contained arsenic concentrations that were roughly proportional to the arsenic intake. The pig receiving arsenic trioxide, however, had significantly less arsenic in the liver, heart, spleen, and blood than the pig fed lead arsenate. Typical of animals ingesting lead, the animal given lead arsenate contained 25.3 ppm of lead in the liver and 192 ppm in bone. Needless to say, animals ingesting lead arsenate receive a double insult, both lead and arsenic.

Cattle and horses reportedly can consume up to 2.04 g of arsenic trioxide daily without toxic symptoms (46).

Arsenicals and Wildlife Losses

Non-Pax Related Considerations

Several instances of wildlife mortality or suspected mortality due to exposure to arsenicals are recorded in the literature. In their study dealing with the effects of arsenic-treated grasshopper bait on chickens, Wilson and Holmes (59) prefaced their publication with a comment on suspected wildlife mortality due to arsenic poisoning. The grasshopper poisoning campaign in Wisconsin in 1934 resulted in the application of more than 10,000 tons of poison grasshopper bait in northern counties. The bait consisted of 100 g sawdust, 1 ounce whey, and 4 g arsenic trioxide which was broadcast over the land surface for consumption by grasshoppers. These workers mention that a number of deer were found dead in the woods and around small ponds, the cause of death not known. Also, an endemic disease appeared among the prairie chickens which so greatly reduced the number over large areas in the grasshopper territory that only a few individuals could be found when the hunting season began. Farmers and sportsmen attributed the mortality to the grasshopper bait.

Lilly (31), experimenting with pheasants, fed poisoned grasshopper bait to this species and concluded that gallinaceous birds are relatively immune to grasshopper poisoning as recommended and practiced. More recent work with pheasants and chickens, however, has demonstrated that these species are notorious for their ability to withstand exposure to many pesticides, and that species vary greatly in their susceptibility to pesticides. Such differences must be recognized and dealt with in whatever assessments of effects of pesticides are attempted.

In the spring and summer of 1952, reports of unusual wildlife mortality in certain areas in the southwestern part of the upper peninsula of Michigan were received by state wildlife personnel (4). These losses (deer, porcupine, rabbit) occurred where paper pulp companies had established test plots to determine the effectiveness of sodium arsenite as a debarking agent on aspen and other tree species. State toxicologists diagnosed the mortality as arsenic poisoning. Animals appeared to be obtaining the arsenic by licking the bark and the ground surface at the bases of the treated trees.

Arsenic acid with an equivalence of 52.3% arsenic trioxide killed 11 deer in Shelby County, Tennessee, in 1971 when used to control Johnson grass on 600 acres (53). One-half gallon of arsenic acid was mixed with 4½ gallons of water and applied by aircraft at the rate of 5 gallons per acre. All deer were found in or near a water source. Please note that the deer found dead in Wisconsin (59) also were found around ponds. Swiggart and his coworkers (59) attribute this affinity for water to a severe gastroenteritis and loss of fluid which commonly attends arsenic poisoning. The Tennessee deer had ingested treated vegetation. Use of the arsenic acid for this purpose constituted a misuse of the material. Delta Brand Arsenic Acid, USDA Registration No. 295-6, is approved for use on Bermuda grass lawns to control crabgrass and Dallis grass.

An undated news release issued by the U.S. Fish and Wildlife Service's Regional Office, Minneapolis, Minnesota, reports a robin die-off in south Minneapolis in which birds contained 200 mg of arsenic trioxide, an ingredient in a commercial weed killer. It is stated in the release that the birds probably ate earthworms containing the arsenic material which had been used to treat the lawns. It is further stated that 5 to 10 mg of arsenic normally would be enough to kill robins.

The Pax Product and Fish and Wildlife

A letter requesting information on any animal mortality, domestic or wild, terrestrial or aquatic, which could be attributed to use of the Pax product was sent to the directors of the fish and game departments in six western states. Selection of the states was on the basis of their close proximity to the Pax formulation plant in Salt Lake City, Utah, and the likelihood of greater use of the Pax product at these locations. Information from the Pax Company on sales volume and distribution of the product was not supplied in time to make use of it in the selection of sites of inquiry. Of the three states responding (Nevada, New Mexico, Wyoming), none had knowledge of animal mortality traceable to the use of the Pax product.

The Pax product as formulated under Registration No. 3234-3 contains 25.11% arsenic trioxide, 8.25% lead arsenate, and 4% ammoniacal nitrogen derived from ammonium sulfate. The recommended rate of application is 40 pounds per 2,000 square feet. At this rate, 181 pounds of arsenic and 43 pounds of lead are applied per acre with each treatment. For perpetual control, instructions call for a yearly application of the same material at a third of the initial rate or a full-rate application every third year.

It is most unfortunate that no definitive experimental studies have been made to obtain information on possible hazards associated with use of the Pax material. Reliance on chance observations is not an appropriate way to evaluate possible hazards to domestic animals and wildlife. Suitably designed studies should be conducted to detect possible hazards if continued use of the Pax product is permitted.

Based on our knowledge of arsenicals, it seems likely that the Pax product is contributing in some measure to the overall environmental load of lead and arsenic; but is is not possible at this time to separate the contribution made by the Pax material from arsenic and lead coming from other sources. Although the two arsenicals in the Pax product are relatively insoluble, a variety of factors will lead to a redistribution of at least some of the material.

Only in extremely localized and straight-line situations is it now possible to relate hazard to use of the Pax product. The loss of horses and cows due to the ingestion of grass clippings taken from Pax-treated lawns is a straightforward example of cause and effect (6). Sublethal poisoning of humans due to inhalation, skin contact, or accidental ingestion also are readily relatable to exposure (statistics on human poisoning assembled for the Pax Review Committee by EPA). The fact remains, however, that no recorded instances of wildlife losses due to the use of the Pax product were encountered, unless the robin mortality in Minneapolis was such an event. Usually, unless poisoned animals remain close to the scene of exposure, extremely expert sleuthing is required to determine the cause of death. Animals poisoned by arsenic do not succumb rapidly, thus providing ample time for movement away from the area of exposure. This coupled with the fact that most wild animals die without detection would make it appear unlikely that poisoned animals would be encountered by chance. There is the distinct possibility that soils of treated areas are rendered so free of the kinds of invertebrates on which birds feed (earthworms, grubs and other immature insects, slugs, snails, etc.) that birds do not forage extensively on treated lawns. This offers an interesting subject for research. Arsenicals are commonly used as soil sterilants and to kill earthworms, snails, slugs, and other unwanted invertebrate animals.

Despite the relatively in situ nature of the more insoluble arsenicals, one would expect movement of a portion of the arsenic and lead in solution and by sediment transport. It seems likely that small ponds and lakes within some treated areas could provide interesting experimental situations. In his studies on the use of arsenicals for crabgrass control in turf, Stadherr (50) mentions that the Pax material accumulated in depressions in an irregular lawn surface and caused dead spots. There is the distinct possibility that high concentrations of the material may be found in these depressions and may constitute a real hazard to dogs, cats, and various species of wildlife that may use such pools for drinking; there is also the chance that children may, on occasion, wade and play in such temporary puddles. This matter should be investigated.

Bioconcentration

Bioconcentration is a term used in the field of pollution ecology to describe the accumulation of a substance in the body of an organism which exceeds the level of intake of the substance as it occurs in the medium in which the animal lives, or in the trophic level(s) which the animal uses as a food source. Some good examples of bioconcentration are to be had by referring to the organochlorine pesticides. Fish may accumulate residues both from the water that surrounds them and from the food they eat (1). Cutthroat trout exposed once monthly to a 30-minute bath in water containing 0.3 and 1.0 ppm of DDT had 4 - 6 ppm in their bodies after 6 to 8 treatments; this represents an increase of 4 - 20 times. Fathead minnows exposed to water containing 0.000015 ppm of endrin had total body concentrations 10,000 time the amounts in the water (37). Woodcock fed earthworms containing about 3 ppm accumulated approximately four times this amount in their bodies in 2 months. Those fed earthworms containing about 3 ppm accumulated about four times this amount by the time of death 16 - 52 days later (51). Starting with a DDT level in the soil of 9.9 ppm, it reached 141 ppm in earthworms and 444 ppm in robins (45), a quite simple food chain that proved to be devastating to the robins where DDT was being used to control Dutch elm disease.

Bioconcentration of Arsenic

In ponds treated by Dupree (18) with sodium arsenite at 4 ppm, the arsenic concentrations in plankton reached a peak of 6,955 ppm arsenic trioxide within 27 days after treatment. In two other ponds that received 8 ppm As_2O_3 in 1955 and 4 ppm As_2O_3 in 1956, plankton reached a peak of

8,200 ppm As_2O_3 within 41 days after treatment. Siseable amounts of arsenic were retained by the bottom muds.

Cope (13) reported that bluegill concentrated sodium arsenite in a few days from a level of 0.69 ppm in the water to 11.6 ppm in adult bluegills.

Macek (34) in discussing biological magnification of pesticides in food chains expresses the view that the majority of the herbicides do not meet the prerequisites for biological magnification, although it appears that inorganics, such as sodium arsenite, could possibly undergo biological magnification.

Isensee et al. (unpublished manuscript) studied the distribution of ^{14}C labeled cacodylic acid (CA) and dimethylarsine (DMA) among aquatic organisms in a model ecosystem and concluded that the lower food chain organisms (algae and Daphnia magna) bioaccunulated more CA and DMA than did higher food chain organisms (snails and fish). "Amounts accumulated indicate that CA and DMA do not show a high potential to biomagnify in the environment. An increase in biomass (primarily algae) over 32 days largely accounted for a gradual loss of DA and DMA from solution. Equilibrium between each organism and the external solution was more important in determining CA bioaccumulation than consumption of one food chain organism by another." It should be noted that these workers make a distinction between the arsenic acquired from the water (bioaccumulation) and the arsenic acquired from the ingestion of the food chain organisms. Many workers do not make this separation in dealing with bioconcentration because it is difficult to determine the manner in which body burdens of a substance have been acquired.

Quite obviously the subject of arsenic bioconcentration needs more study. On the strength of what is known on this subject to date, it appears reasonable to subscribe to the idea that arsenic does bioconcentrate in some animals. Some of the arsenicals are quite persistent in soils; some are rather readily taken up and stored by plants and animals in fairly good quantity; arsenic is readily recycled; what few species of animals have been studied to date, there is evidence of some differences in sites of storage, degree of binding in tissues and organs, and rates of excretion. These characteristics coupled with the investigation of a variety of species and ecological situations yet untouched will undoubtedly bring to light a number of deviants.

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PAX ADVISORY REPORT, SECTION ON TOXICOLOGY

All arsenical compounds are capable of causing injury to the animal or human organism. What is not generally understood is the variation in the toxic potential of the many compounds contained within the arsenic spectrum. As a rule, inorganic arsenicals are more toxic than the organics and trivalent forms are more toxic than the pentavalents. Notwithstanding the well established poisonous nature of arsenic, both trivalent and pentavalent forms have been used as therapeutic agents in the treatment of syphilis and certain parasitic diseases with beneficial effects. Modern application in this regard has replaced arsenicals with more efficient and less toxic medicaments.[1,2]

TOXICOLOGY

Toxic agents induce their effects by physical or by chemical or physiologic (enzymatic) mechanisms or sometimes by a combination of the above. The two fundamental considerations are the action of the intoxicant on the host and the reaction of the host to the intoxicant. Allied factors that influence toxic response are: route of entry, dose, particle size, solubility of the material, and individual susceptibility.[3] Just how trivalent arsenicals exert their action on the animal organism is not completely understood, but certain views are held. For example, it is believed that one of the ways trivalent arsenicals (certain forms more than others) exert their toxic effect is by inhibiting the pyruvate oxidase system which is essential for energy transformation within cells. There is also experimental evidence that certain enzymes, d-amino oxidase, 2-glutamic acid oxidase, monoamine oxidase, and transaminase are inhibited in various degrees by trivalent arsenicals; whereas other enzymes are inactivated. Those enzymes which contain sulphydryl groups are highly susceptible to the action of arsenic.[1,4,5] Wadkins states that arsenic is also capable of uncoupling oxidative phosphorylation in liver mitochondria, thus interfering with ATP formation.[5,6]

Both of the arsenicals contained within the herbicide product being reviewed are well known compounds to industry and agriculture. Arsenic trioxide is a by-product of nonferrous arsenic containing ores and is encountered in certain insecticides, weed killers, poison baits, glass making, enamels, alloys, taxidermy, wood preserving, and metal smelting. Considerable exposure to this compound can occur in the smelting of lead, copper, cobalt, tungsten, and gold ores containing metallic arsenic.[7] Smelter workers can experience absorption of arsenic by way of inhalation, ingestions and skin contact as can be demonstrated in elevated urinary values for arsenic. Rarely, however, do these exposures produce signs and symptoms of an acute arsenical intoxication. The major problems reported in such workers by Pinto et al[7] in the United States and Holmquist[8] in Scandinavia have been related to manifestations ranging from mild to moderate dermatoses, including primary irritation of the mucous membranes of the nose, mouth and eyes. Nasal perforation is not unusual. Holmquist's most extensive and careful study of the cutaneous lesions caused by arsenic trioxide in industry showed that contact dermatitis was by far the most common ailment observed among these workmen. He described cutaneous changes as eczematous dermatitis, toxic dermatitis and combinations of the above. Birmingham et al in 1964[9] observed similar skin lesions, including ulcerative changes of skin folds, among the workmen and several children who lived in a western mining community where arsenic trioxide from the smelter became a plant and community pollution problem.

Skin changes caused by arsenic trioxide are largely primary irritant in nature, with a small percentage resulting from allergic hypersensitivity.[9] Industrial experience with arsenic trioxide has been generally good because of hygienic practices as ventilation controls, daily change of work clothing, use of respirators, and daily showering. Even in plants with poor hygiene, acute intoxication from inhalation or skin contact with arsenic trioxide is rare.[10]

Acute intoxication from arsenic trioxide generally arises from accidental or purposeful ingestion. Clinical signs, symptoms and severity usually parallel the dose, but individual susceptibility can vary. The affected animal or human, depending upon the dose, displays a variable time of onset of gastrointestinal signs characterized by dryness and irritation of the oral cavity, difficulty in swallowing, severe abdominal pain, nausea, wretching and vomiting, followed by profound diarrhea similar to that observed in cholera. Direct action of the intoxicant upon the blood vessels in the bowel induces blood-streaked stools, severe dehydration, exhaustion, and shock. The basic lesion is associated with increased permeability and dilitation of the small arterioles and capillaries. Toxic dosage of trivalent arsenic varies considerably, but it is highly dependent upon solubility, particle size and, obviously, the amount ingested. Chronic arsenical intoxication can be expressed in a number of clinical displays which include gastrointestinal and neurological effects, kidney and liver damage, and melanosis or pigmentation of the skin.[1, 2, 4, 11] These problems have been surprisingly uncommon in industry, even in plants where preventive practices were absent.[1, 7]

As to the toxicity assessment of the product in question, both arsenic trioxide and lead arsenate have a lower order of solubility. Nevertheless, they are capable of causing toxic effects if sufficient exposure occurs. Sollman[12] quoted 0.2 to 0.3 grams of trioxide as a fatal dose. Thienes and Haley[13] refer to the fatal dose of arsenic trioxide as approaching 0.1-0.2 grams. Done[14] has correctly pointed out the lack of precise toxicity information associated with arsenicals in general and trioxide in particular. For example, the LD-50 information may range from 9 to 39 to 500 mg./kg. Hueper and Payne[15] reported no significant adverse effects in rats and mice fed 34 p.p.m. of arsenic trioxide in water for a period of 24 months. Hatch and Funnel[16] state that in cattle the oral lethal dose of arsenic trioxide is 15 to 45 grams, whereas the lethal dose of sodium arsenite is 1-4 grams. Calvary et al[17] performed feeding experiments using dogs to study the chronic effects of lead acetate, lead arsenate, and arsenic trioxide. He fed the lead acetate at three levels, 12.8, 38.4 and 64 mg. of added lead per kg. of diet; lead arsenate was fed at 64 mg./kg. of diet; and arsenic trioxide at two levels, 26.8 and 107.5 mg./kg. of diet. All of the dogs fed arsenic diets survived. Of 20 dogs fed on different levels of lead, 15 died of lead poisoning.

Done and Peart[14] compared the toxicity of sodium arsenite to that of arsenic trioxide and an organic arsenical administered to young rats. This work was done partly because of the lack of information dealing with comparative acute toxicity data of various herbicides in experimental animals. They ascertained from their work the following LD-50s: Sodium arsenite - 42 mg./kg., arsenic trioxide - 385 mg./kg., PAX product - 156 mg./kg., lead arsenate - 344 mg./kg., and calcium arsenate - 100 mg./kg. These results are in general agreement with the arsenic poisoning information compiled from (1) The National Clearing House for Poison Control Centers; (2) Pesticide Regulation Division accident files; (3) Summary of 62 pesticide accidents investigated (1963-1966) by Plant Pest Control Division. From 1939 to 1967 there were 184 cases involving 202 victims of which 39 fatalities were attributed to arsenic. Twenty-four of these were due to sodium arsenite; seven to arsenic trioxide; one to lead arsenate; and seven to other undetermined arsenical compounds. Three of the 39 cases were suicides. All of the poisonings were due to ingestion. None of the fatalities was attributed to the product in question or to the more potent herbicides marketed by the company at that time. In Arsenical Poisoning Informational Data,[19] January '68-September '72, compiled by the Accident Investigation Section of the Office of Pesticide Programs (October '72), 29 deaths were reported. During this same period, PAX Crab Grass Control was the cause of fatal ingestions in five horses, five cattle and bees. The cattle and equine fatalities resulted from having fed the animals lawn clippings which had been treated with PAX crab grass arsenical. From the data presented by the Poison Centers, including Dr. Done's material from USDA, it appears that PAX has not caused any human fatalities, but it has been involved in nonfatal illness of humans and several deaths in livestock. The 1969 episode involving the death of five horses which had eaten PAX treated grass has been fully described by Buck in a communication obtained for this committee.[20] Postmortem findings in two of the horses showed arsenic in the liver, kidneys and stomach. Postmortem findings were also described in cows which had eaten grass clippings from a lawn treated with PAX some five to six months earlier. Further investigation was conducted by Buck in which PAX treated grass clippings were fed to a young calf which died in 27 hours. The arsenical content of grass clippings in the liver, kidney and rumen were chemically validated in the laboratory. Two rabbits fed grass clippings from a PAX treated lawn also suffered fatal toxic effects and showed arsenic in the liver, kidney, stomach contents and feces.

A critique of this report submitted by B. R. Ellison,[21] Research Director of the PAX Company, took strong issue with several statements made in the Buck report. In short, the company did not accept the diagnosis of arsenical intoxication in the horses examined at autopsy.

HAZARDS TO USERS AND APPLICATORS

Most sources of toxicologic data agree that arsenic trioxide and lead arsenate have a lower toxic potential than several other arsenical compounds. Up-to-date evidence of the effects of lead arsenate in humans was presented in a cohort study by Nelson et al[22] involving orchard workers in Wenatchee, Washington, with variable lead arsenate exposure indices. The study was conducted on 1,231 individuals who had participated in a 1938 morbidity survey by the USPHS of the exposure effects of lead arsenate spray used on the apple trees. Classification included spray exposure, duration, age and sex. The orchardist group had the highest exposure; a consumer group had no direct exposure. A third group had intermediate exposure. Over 97% of the original group was located. For all study members combined, the standard mortality ratio was 70%. The cohort group experienced less mortality than the Washington average.

The product in question is considerably lower in arsenic content than was the case with the other PAX products which poisoned the animals and which recently have been withdrawn. None the less, the present product is quite capable of providing toxic insult and does constitute a hazard where children and pets may have access to it if stored in garages, basements, out buildings, barns, etc. The actual application of PAX Crab Grass Control as prescribed does not constitute a formidably toxic potential because inhalation and ingestion factors would be minimal. Body contact while applying the mixture should be avoided, but systemic toxicity from body contact is not probable. Cutaneous effects as contact dermatitis, irritant folliculitis, pyodermas, and ulceration of the body folds, including the finger and toe webs, could occur if the trioxide is not removed from skin a few hours after application. Twenty-five percent arsenic trioxide is entirely capable of producing skin ulcerations, but the chemical must remain on the skin for several hours. This irritant propensity is enhanced by the action of sweat.

When the applicator waters in the material, some contact through displacement onto the applicator's clothing and footgear could occur, but this does not appear to be a major problem.

HAZARDS TO THE PUBLIC

It is unlikely that treating plots of land with PAX Crab Grass Control product would cause systemic toxicity from airborne pollution with arsenic. It is conceivable that adults or children walking barefooted in the freshly treated plot and failing to wash soon thereafter could develop primary irritant ulcerative effects on the skin, particularly between the toes. This situation was observed in several children in the arsenic pollution episode in Nevada by Birmingham et al.[9] Further relevant reference has occurred in reports from the company under the term

"PAX paws," as noted in dogs. Such ulcerative phenomena probably result from the trioxide granules being held in the crevices of the paws. It is conceivable that a pet could be poisoned from licking its contaminated feet, however, the amount involved is small indeed and when this fact is added to the low solubility and large particle size of the arsenic it diminishes the chance of systemic effects by this route.

The possibility of children ingesting candy or food which has been dropped on a treated plot does exist. However, the amount of arsenic which would adhere to an edible material seems insufficient to warrant great concern.

If it is true, as has been stated, that animals are attracted to arsenic then it is possible that they could lick or eat the freshly treated grass with possible ill effects. It has also been stated that the bovine species is attracted to soil containing arsenic.

COMMENTS

A review of the literature dealing with arsenic, its effects on pathophysiology, its toxicologic and clinical signs in the animal and human organism has been made. There seems to be little question that the two arsenicals used in the PAX Crab Grass Control product are capable of causing toxicologic response. However, data from Poison Control Centers, the U. S. Department of Agriculture and other informational centers have not validated any human fatality from this product. Animal fatality allegedly has resulted from feeding PAX treated grass clippings to the livestock. Animals fed PAX treated lawn clippings under controlled observations did succumb to arsenical intoxication.[20] The potential hazard to children and pets associated with storing such products around the house is significant. The possibility of applicators and the public developing ill effects from this material are remote, though not impossible. It is conceivable that children and adults could develop irritant reactions on the skin by walking barefoot or by lolling around on treated grass while wearing a minimal amount of clothing. It is already known that dogs can develop ulcerations of the paws because of the action of the arsenic trioxide, but there was no evidence in the literature that the dogs died from having experienced such exposures. As to the question of young children ingesting candy or food which had been dropped on freshly treated plots, it is unlikely that enough arsenic would be taken in to be of serious consequence. A remote hazard should be recognized in the case of the child afflicted with pica, since he might ingest soils containing high amounts of the freshly treated area.

After reviewing Dr. Buck's excerpt of his paper dealing with the crab grass control product, one is convinced of his strong feelings concerning arsenic herbicides in general and the product in particular. The episodes he described were unfortunate and, indeed, uncalled for, but they did happen. In Mr. Ellison's rebuttal, I share his criticism concerning the analytical data presented by Dr. Buck, but found it difficult to readily agree that the animals probably died from an arsenical other than PAX.

As far as safety and health is concerned, it would be preferable if all arsenicals were used under stricter controls. Even that system, however, would not remove all of the problems associated with economic poisons. It is unfortunate that we have to rely on potentially lethal materials to manage crab grass. However, since their use is permitted in certain areas of the country and in view of the scientific evidence available, I find it unwarranted to selectively eliminate this product for the purpose intended while permitting use of other agents with equal or more toxic potential.

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5-8-73
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Discussion

Despite the fairly exhaustive reading and literature research conducted in our brief span of study of the PAX Company problem, and considering arsenic history and attitudes toward "arsenic", it would be surprising if the committee had reached unanimous agreement. In the first place, it is very clear that the arsenicals involved are quite toxic to animals and to man if injested in sufficient quantity. There is also the tendency to enact laws and regulations to try to protect individuals against the misuse of pesticides and other agricultural chemicals. The fact that tolerances for arsenic in foods seem to be almost a part of our culture must also be taken into consideration. On the other side of the argument, there stands the strong evidence that inorganic arsenicals play little understood positive roles in biology, possibly as a catalyst for phosphorylations in animals (30)* and in photophosphorylations in plants (31). If ostracized, there will be little or no incentive to learn more of the benefits inherent in their safe uses.

Although used successfully as a persistant soil sterilant by Crafts and Rosenfels (32), without hazard to wild or domestic animals, the value of arsenic trioxide in horticulture did not end there. The surprising discovery was made separately that lead arsenate and/or arsenic trioxide at somewhat lower levels had remarkable value to repress the germination of crabgrass seed when used at the soil surface. Reports by McNulty and Rhodes (33) brought this to the attention of the Utah Cooperative Association, leading finally to the development of a patented combination of arsenic trioxide with lead arsenate (3). The Utah Cooperative Association then established the PAX Company to market this product, which went through many experimental improvements, all registered for use by the U.S. Dept. of Agriculture. PAX Three-Year Crabgrass Control products were sold mainly for use on home lawns and were handled by hardwear and garden stores. In some cases PAX found use on golf courses. That the product appeared trouble-free and satisfied its buyers for about 20 years is evident from the testimonials seen in Appendix 1.

Subsequently, calcium arsenate came into widespread commercial use for the control of crabgrass and *Poa annua* on golf courses. The

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* For references, see Section 1.

Chipman Division of Rhodia marketed the product Chip-Cal^R along this line. Their investigation and resulting action assured continued use of tricalcium arsenate on golf courses.

In 1970, New York, Iowa, and other states enacted legislation providing for the licensed application of inorganic arsenicals. This action was welcomed by Rhodia and used as a means to advance the sale and safe use of their products. Meanwhile, the older PAX Company products, based on arsenic trioxide and lead arsenate, fell into disfavor in many states, particularly in Iowa where the unfortunate accident recounted by Buck (2) had occurred. Governmental efforts to inveigh on the PAX Company to remove their product from the market in 1970-71 were overcome by legal defensive action on behalf of PAX, supported by the safety evidence provided by Done and Peart (1). It then became painfully evident that 20 years of safe usage did not obviate all possibilities for misuse of the product, particularly in the chance event of the feeding of grass clippings to animals. A brief survey of turf management experts acquainted with the use of calcium and lead arsenates on golf courses disclosed that none were aware of poisoning in animals or humans stemming from the recommended use of such products. This is somewhat surprising in that calcium arsenate is somewhat more toxic than PAX.

That the licensed application of such toxic pesticides will reduce all possible hazards to the public can hardly be denied. On the other hand, the removal of responsibility for individuals to think for themselves and to act for the welfare of all, is the essence of democracy. It costs far less where people manage their own affairs in such matters than where government intervenes. The views expressed by Drs. Kearney and Woolson, i.e., "...I don't think we should regulate the ability of citizens to use a product safely." (Appendix 2) seem to be the crux of the matter.

The efficacy of PAX lawn care products is evident from the 20-year sale of the product; also from unsolicited testimonials from homeowners and golf course superintendents (Appendix 1.). The PAX products are now permitted or marketed, so far as our records show, only in certain western states. Such states have predominantly alkaline soils, quite different from those in the East. To raise and maintain good turf in many of the western states is a more difficult problem than in the East. For this reason and others, consideration might logically be given to assigning authority to each state regarding the safe uses of inorganic arsenicals for turf management. It should be recognized, however, that such household items as aspirin, boric acid, oxalic acid, gasoline, guns, knives and automobiles are at least as injurious when misused.

Recommendations

The primary recommendation is for further research:

1) To establish better knowledge of the precise values of inorganic arsenicals for turf improvement.
2) To establish ways to minimize hazards from such uses.

Recommendation is made to support research toward better understanding of the fundamental role of arsenic in plant biology, its relation to phosphorus and nitrogen metabolism, particularly to conservation of essential resources of phosphorus. In view of the long and unique, but little understood value of lead arsenate as a pesticide, catalyst for maturation and improved quality of fruits, and for turf management, we urge further research on its safe uses.

In view of the record, it is recommended that registration of the single PAX Company Three-Year Crabgrass Control product now registered be continued in registration with the Department of Agriculture and/or the Environmental Protection Agency. It is further recommended that those charged with turf management and regulatory controls of pesticides in each state again review the overall problems relating to the safe uses of PAX Three-Year Crabgrass Control, calcium arsenate, and lead arsenate products for turf improvement. Whereas licensed application seems appropriate for such products for commercial turf, such as for golf courses, public parks and grounds, imposition of such restraint seems inappropriate for home-owners. More prominent label warnings against feeding arsenical-treated grass clippings to animals should suffice to reduce hazard of the PAX product for home use.

AFFIDAVIT

State of Utah)
County of Salt Lake)

B. R. Ellison, being first duly sworn, deposes and says:

It is my understanding that the Committee would like me to furnish them in affidavit and illustrated form as much of the information as possible which Dr. Frost asked me for at the time of his recent visit with me in Salt Lake. Therefore, this affidavit will contain a rather general discussion on some of the technical aspects of Three-Year Pax Crabgrass Control, the arsenicals which it contains, and their behavior on the soil. From time to time, in our discussion, a comparison was drawn with other arsenicals, particularly calcium arsenate, sodium arsenite, and lead arsenate.

Dr. Frost wanted information about country clubs and golf courses which have treated their entire fareways. Also, he wanted to know if we had any affidavits or recommendations from golf course superintendents. We have such information and it will be discussed in the affidavit.

Dr. Frost asked me specifically if I had some areas that showed the encouragement of desirable turf grass plants as mentioned in Dr. Richard Stadtherr's thesis. I was able to show him such a plot.

Dr. Frost selected four pictures from a number of my slides which I showed him which he felt should be contained in a report to the Committee. These are therefore attached as exhibits to this affidavit with an explanation of what they show.

Dr. Frost asked for explanation of what might be done if a person treated an area with PAX Crabgrass Control and then decided that they wished to grow something else besides turf in the area. We discussed this and the discussion will be contained in the affidavit.

GENERAL DISCUSSION OF A TECHNICAL NATURE CONCERNING PAX THREE YEAR CRABGRASS CONTROL, THE ARSENICALS CONTAINED IN IT AND THEIR BEHAVIOR IN AND ON THE SOIL.

It is a well known fact that certain crop lands have been taken out of production by the repeated use of arsenicals used as insecticides. Probably most famous of these damaged soils occur in the orchard areas of the northwest and some cottonlands in various parts of the country. It is characteristic of these soils that arsenical can be found at considerable depths in the soil, well into the root zone of the plants, and a considerable reduction in yield is characteristic of these areas and attributed to the high arsenical content of the soil. The important thing to realize in these soils is that the arsenical did not migrate to these great soil depths by itself but rather was cultivated into the deeper layers of the soil. Arsenical compounds, like phosphates, are very tenaciously bound by various fractions of the soil, some of which are clay particles and organic collatable materials. Some turf areas, particularly those that are not receiving adequate amounts of nitrogen fertilization will accumulate an abnormal amount of thatch or litter layer. Such areas are quickly recognized on the golf course because of the watering problems they present. This thatch layer forms a very efficient water barrier. Fairways with such areas have serious problems of water management. While the homeowner may not recognize the condition as readily as the golf course superintendent, it nevertheless exists in some home lawns. Obviously, if such an area resists the penetration of water, it also resists the proper washing in of any pesticide material, particularly PAX Three Year Crabgrass Control. In such an abnormal lawn, a fairly large percentage of the arsenical will become fixed in the thatch layer and never reach the mineral soil at all. A PAX Three Year application in the Fall on such a lawn would not be washed in. Rather, it would become bound on the decaying thatch layer. Virtually the entire PAX application might very well be removed the following Spring if the area were power raked. It is likely that a sizeable percentage of the PAX application would be removed even by close mowing with a vacuum type rotary mower.

The ultimate fate of arsenicals placed on the soil surface of a stabilized soil, such a growing turf, is quite different from that of an arsenical which is applied year after year on cottonland or in orchards. For one thing, a rate of arsenical, tremendous by standards of PAX Crabgrass and Soil Pest Control, is sprayed on the fruit trees and plants not once in three years but several times in one season. Although the application is made to the trees and cotton plants, most of it ultimately reaches the orchard floor or soil. Later it is plowed and cultivated under usually too rather substantial depths.

Arsenicals applied to a stabilized soil are usually found within the first half inch of soil with by far the greatest part of it within the first quarter inch. If the management of this soil promotes a good deal of growth of micro-organisms, as would be the case with a turf area, there are a number of microorganisms that are normal and common members of the soil microflora that produce exogenous enzymes which catalyze the production of volatile arsenical compounds which pass into the atmosphere. It is characteristic, as determined by many soil samples run by the PAX Company for a number of years, that characteristically approximately half of the arsenical is dissipated in the first year after application.

It is generally concluded by soil chemists that the soil chemistry and behavior of arsenicals in the soil is similar to that of phosphorous compounds and that it is likely that arsenicals and phosphate compounds compete for positions on the colloidal micelles of the soil.

It is known that arsenical compounds exert a distinctly stimulatory affect on certain crops, many of them grasses. Dr. Frost and I discussed the possibility stimulatory affect might possibly be due to the arsenical compound making available a certain amount of phosphorous as a result of the arsenical displacing the phosphorous on the soil colloid. I have no information on this, but I do have some studies on the reverse situation. It occurred to me that if there was a mutual competition between phosphorous and arsenical for a position on the clay particles or other colloidal particles in the soil that an application of a phosphate material to an area that has previously received an application of arsenical might result in arsenical damage to the turf or perhaps increased weed control simply as a result of making more of the arsenical available in the soil solution as a result of the phosphate displacing it from the soil colloids. Both field and laboratory studies are carried on in an attempt to investigate this possibility.

In the field, heavy applications of arsenic trioxide were made to a turf area, washed in, and left for a period of one month. These plots were in three replications. In a month after the initial application, an application of diammonium phosphate was made to one half of each plot and washed in. The rate of arsenicals applied to the turf was arsenic trioxide at the rate of 20# per 1,000 square feet. The arsenical was applied alone, without the fertilizer and insecticidal materials which characterized the product PAX Three Year Crabgrass Control. I think the reason is obvious. The diammonium phosphate was selected because it is a very soluble phosphate. It was applied at a rate equal to 1# of P_2O_5 per 1,000 square feet. No difference could be detected through the entire season between the two halves of the three replicated plots.

In the laboratory, a screened, sandy loam, soil was shaken in flasks that had a saturated solution of arsenic trioxide (actually arsenious acid). These soils were filtered out of the flasts and leached with saturated solutions of diammonium phosphate. We could detect no liberated arsenic in the leachate.

Utah soils are generally characterized by having high levels of phosphate. All turf soils in the country were for years fertilized most characteristically, and improperly, with a 6-10-4 fertilizer. Most old lawns contain a very high phosphate level in the soil, no matter in which part of the country they occur, as a result of this improper type of fertilization which was charactestic earlier.

Golf courses, likewise, were generally heavily overfertilized with phosphate containing materials. Phosphate levels in the soil on most golf courses are fantastic. If there was any great antagonism between arsenic and phosphate, turf areas should be the last place on which a company should venture with an arsenical herbicide. Indeed, competitive companies marketing calcium arsenade or lead arsenade for the control of crabgrass or other weeds suggest that their product may not work if the soil contains rather large amounts of phosphates. We do not seem to have inhibition of our combination of arsenicals as a result of high phosphates in the soils. A Denver golf club, Green Gables, treated their fairways with great success with our product, PAX Three Year. The product was very successful in spite of the fact that soil tests showed that the fairways averaged 1,000 pounds of available P_2O_5 per acre. Thirty pounds of available P_2O_5 for a phosphate loving crop like alfalfa, is marginal. If available P_2O_5 is below that, most state soil labs would recommend fertilization with phosphate, but if it was substantially above thirty pounds per acre, they would not.

Remarkably enough, for areas that have suffered crop damage from the application of arsenicals, zinc chelate seems to be more effective in counteracting the effect of the arsenical than phosphate applications.

Arsenicals placed on crop lands that are cultivated and plowed every year may constitute a serious hazard to such lands. This is largely because the arsenicals that are applied and later plowed down to six to eight inches depths, have little chance of escaping. Certainly complex chemical reactions occur with these arsenicals and some volatile arsenicals are produced by micro-organisms but when the arsenicals are deep in the soil, the volatile arsenical is usually recaptured in some cycling chemical reaction and it does not escape. A further difference between the application of an arsenical to the surface of a turf soil as opposed to a cotton crop or an orchard floor is that the micro-organism activity is extremely high in the superficial layers partly because both moisture and nitrogen are usually readily available at the superficial layers. This, of course, promotes great micro-organism activity. In the deeper layers of the orchard floor or the cotton field that has plant detritious plowed under with the arsenical until the nitrogen may be extremely scarce, water relatively scarce, and consequently micro-organism activity is extremely low.

Dr. Frost ask me what would happen if a person treated an area with PAX Three Year Crabgrass Control and then changed his mind and decided to plant something else. Of course a number of factors would be involved in this situation. Plants vary remarkably in their vulnerability to this combination of arsenicals. We regularly plant and establish bluegrass seeds right in a PAX application. I don't think that it is necessary to speculate on the possible fate of agricultural crops since our product is specifically designed for established lawns. There is some indication that the consequences would not be very serious since a good deal of modern suburbia has moved out on to the truck garden farms of Long Island and such similar areas which were regularly treated with arsenicals. In these areas they have managed to establish landscaping, in fact very beautiful landscaping, with the minimum of difficulty. There is approximately 20% of elemental arsenic in PAX Three Year Crabgrass Control. At a recommended rate of 20 pounds per square feet we would apply approximately four pounds of elemental arsenic applied per thousand square feet. Soils laboratories generally use the figure of 100 pounds per cubic foot for soil. If we could persuade this changable fellow to turn his soil over to a depth of 6 inches, and that would not be unreasonable, we would end up with approximately 80 parts per million of elemental arsenic in the soil which would constitute the major root zone of most ornamentals. Many soils that grow normal vegetation have higher natural rates of elemental arsenic than this.

Certainly if we were considering fertilization and discussing the application of a phosphate rather than arsenical we would not assume that 100% of the phosphate would be available to the plants. Phosphate is quite regularly fixed in many soils in forms that are unavailable to the plant. Some soils are so notoriously efficient at fixing phosphate that it is almost impossible, at least from the economic point of view, to fertilize them heavily enough with phosphate to grow a crop that does not show phosphate deficiency. Certainly all soils fix some of the phosphate that is applied. We find this to be the case with the arsenicals in our product as well. Two areas in our distribution area have some soils that are notoriously efficient in fixing arsenicals. One is the Arcadia area (a suburb of Los Angeles) and the other is in the vicinity of St. Joseph, Missouri. In these areas, even at high rate, we get no control of crabgrass. The dealers in these areas are alert to the situation and so little difficulty develops. When a customer buys the product in these areas and reports poor control, we refund his money with an apologetic letter pointing out that the fertilizer in the product did at least justify the spreading of the product. I do not understand the mechanism of the fixation of the product in these areas but at least the phosphate levels in the soil samples that we have had determine from these areas do not run anywhere near the levels found on some golf courses where our product has been particularly successful.

GOLF COURSE APPLICATION

Dr. Frost was particularly interested in seeing some golf courses that had treated their complete golf course with our product. Incidently, at this point I should make it clear that we do not recommend this product on golf greens. It has been used successfully by a number of superintendents and Dr. Cornman of the New York State College of Agriculture at Ithica had a demonstration application at Nassau County Parks Golf Course. His application of our product, along with Dacthal and a number of other leading crabgrass controls stole the show. Nevertheless we simply do not wish to be involved in the liability that could result from the misuse of our product on a golf green. We are talking about approximately $10,000 worth of turf. This turf is generally abused in every way conceivable. It is a virtual miracle for the golf course superintendent to bring it through the season still alive. When a green dies, the superintendent usually loses his job. To avoid this, a superintendent cannot be blamed for throwing suspicion toward the most recent pesticide he applied on his greens. We do not wish to be in that position but we stand completely ready to back our product on fairways. Our company does not have a golf course program. Nobody in the company calls regularly on golf courses and we do not intend to. A company must have a whole line of golf course supplies to make it profitable to devote the time and manpower necessary for a golf course sales program. We do not even package our product in a convenient size to be used on large areas of turf. Remarkably enough, each sale to a golf course has resulted from experimental applications made either by the PAX Company or, in many cases, research men from the various State Experiment Stations.

The first complete golf course sale resulted in exactly that way. Dr. Victor B. Youngner and Tosh Fuchigami of the Department of Forticulture and Ornamental Horticulture at the University of California at Los Angeles made some crabgrass control applications of an experimental nature on the 15th fairway of the Bel Air Country Club in Beverly Hills. The results of their application was published in the Southern California Turfgrass Culture which was the official organ of the Southern California Turfgrass Council. It is attached, marked Exhibit I. As you will note, the arsenical content of the product at that time was the same as it is today. It differs from the present product in two respects. One is that the insect control was amplified with Chlordane at that time rather than Heptichlor and two, the fertilizer grade was 7-0-0 rather than todays 4-0-0. You will note that the rates were excessively high, 3.33 pounds and 2.5 pounds per hundred square feet respectively. These plots were replicated three times and the general effect on all six plots was slight turf damage followed by quick recovery. This was fertilizer burn from the excessively high rate of fertilizer applied. It is generally a rule among turf men, and a very good one, that no more than one pound of actual N be applied per thousand square feet at any one time. At the high rate, they were applying 2.33 pounds of actual N per thousand square feet. It was at this point that I was brought into the PAX Company. My first contribution was to lower their fertilizer grade to a more acceptable level. Nevertheless, these were rather historic plots because they remained for a period of four years showing almost perfect crabgrass control after all of the other plots have completely faded away. As a result of this, Bel Air Country Club contacted us for a price to treat all eighteen of their fairways. We supervised their application and it was so successful that the following year three of the prestige clubs in the neighboring area in Los Angeles treated their complete fairways. Testimonials of the superintendents of these clubs are included marked Exhibit II.

Golf courses are usually interested in our product for one of two things. Either crabgrass control, as was the case with the Los Angeles clubs, or Poa annua control. Remarkably enough fairways that are infested with Poa annua are very seldom infested with crabgrass, at least this is true in the West. Poa annua in the West is a particular problem in golf courses because since we regularly irrigate our turf areas, the Poa annua is seldom but under enough stress of heat and moisture to die. Consequently from the Rocky Mountain states West through California and the Northwest, Poa annua is not a true annual, but acts as a weak perennial. Control of *Poa annua* in the Middlewest and generally the Atlantic seaboard, at least the Southern part of the Atlantic seaboard, is a relatively easy matter because in late July it all dies. It is a Winter annual in such areas and germinates again in late August or early September. There are several preemergents that will prevent its re-establishment. Some of the best of these are Betasan, Dacthal or Benefin. These kill the seedling shortly after germination. In those states in which *Poa annua* acts as a weak perennial, preemergence controls are not successful. A post-emergence control is required. It is usual on *Poa annua* infested golf courses for the *Poa annua* to be by far the dominate species. Even though the course was originally planted to perennial turf, the *Poa annua* rapidly replaces it. In some sections it comprises 100% of the turf and it is not unusual for whole fairways to be in excess of 90% *Poa annua*. Since *Poa annua* is shallow rooted and subject to a number of fungus diseases as well, such fairways can literally die overnight.

Obviously it would be desirable on such courses to get rid of the *Poa annua* but the situation is something like riding a tiger. It is impossible to get rid of the *Poa annua* all at once because that would leave a bare fairway -- a situation that would be totally unacceptable to the golf course membership. I am convinced that PAX Three Year is the only product that can take out *Poa annua* so slowly on such fairways that the perennial turf can expand and keep up with the disappearing *Poa annua* so that at no time do we leave the fairway bare. The whole process takes approximately three years and is a matter of understanding the nature of both the perennial turf; the weed, *Poa annua*; and the product, PAX Three Year Crabgrass Control. I, or someone else from the company, most regularly supervises the application on fairways that are as heavily infested as this.

Such fairways are to be found on the Salt Lake Country Club which I took Dr. Frost to see. This was the first golf course, in which the fairways were almost totally composed of Poa annua, that treated all 18 of their fairways. Again, no attempt was made to sell this club the product. The club decided to buy as a result of several test plots which I placed on their 8th fairway. Since the entire club was treated approximately 6 years ago, their fairways are no longer composed almost completely of *Poa annua* but *Poa annua* is beginning to return to the fairways. The club is now in the process of retreating some of the more heavily infested fairways. Dr. Frost saw treated fairways into which the superintendent had drilled Merion Bluegrass seed because there was not enough perennial turf grass left to give cover when the *Poa annua* disappears.

As a result of the sucess of the treatment at the Salt Lake Country Club, all the other country clubs in our vicinity that had any kind of budget to work with treated their fairways. Amoung these are Hidden Valley Country Club, Willow Creek Country Club, Oakridge Country Club and Riverside Country Club. Several country clubs in Denver have treated. The first one was the Green Gables Country Club which treated their entire 18 fairways. The result of these applications is given in reprints from the June 1971 "Rocky Mountain Golf Course Superintendents Reporter". This is the official organ of that chapter of the Golf Course Superintendents Association of America. The superintendent of Green Gables Country Club, Ben Struempf, wrote the article. Although his understanding of the nature of the operation of the product is somewhat faulty, his spirit is good. The fact of the matter is they turn their 18th fairways from a miserable mess of *Poa annua* to 18 fairways of good perennial bluegrass. In this course, they again seeded directly into the PAX application with the bluegrass seed because they did not have enough perennial grass to give any cover when the *Poa annua* went out.

PHOTOGRAPH OF RESULTS

From a number of slides which I showed Dr. Frost, he selected four which he thought it would be desirable for the committee to see. I have had them reproduced in black and white. I would have had colored prints made from them, but they would have taken longer than we would had available. Colored prints could be made available to the committee later if they so desire them. The first photograph (Exhibit IV) is a picture of a plot which I took Dr. Frost to see. This plot is ten years old this spring. It was not put on by anyone in our company. It was put on by the Extension Agronomist, Lewis Jensen. A number of other materials were used in this turf area and some of them gave good control the first year, of the crabgrass. However, their was no control the second year. I have heard that it is the object or at least it was the object of the registration section when it was contained in the USDA, to remove all long residual hermicides from the market. If long residual is a sin, we are certainly guilty. Our name implies it, our results confirm it. I repeat, the plot that Dr. Frost saw was ten years old. At the time, our grass was just beginning to break winter dormancy and green up. The location is one of our city parks and the turf care in the city parks is virtually non-existent. The so called turf in the city park consists mainly of weedy grasses, dandelions, crabgrass, etc. Of course, at this time of year it is too early for crabgrass to be showing. Crabgrass germinates in our area most regularly about May 20th. Nevertheless the plot was completely obvious because of the superior nature of the turf. The grass was practically all perennial bluegrass in the plot whereas the rest of the area, which had been treated with various chemicals at the same time our plot was treated was covered with a so called turf consisting of a weed patch with the sprinkling of various kinds of grasses, some of them very undesirable. I showed Dr. Frost this plot in response to his request to see an area in which the PAX seemed to promote the growth of good grasses as Dr. Richard Stadther claimed in his thesis. I think the demonstration was convincing and I can repeat it in any part of the country. The picture was taken in late July, three years after the original application. Nothing was ever done to this turf area in the way of retreatment of fertilization. I am pointing to the extremely straight line of crabgrass control which exists even after three years. This is another plain indication that the arsenicals are strongly bound to the soil and do not move. The crabgrass in the foreground is so luxuriant it looks almost like good turf. It has not yet begun to seed. Indeed, it is so luxuriant that it is covering up a lot of the other weeds that are present such as dandelion, etc. Our product by actual count gets approximately 85% control of dandelions and it can be minipulated to give 100% control. Nevertheless this does not appear on our bag for the simple reason that if you tell a customer that you give dandelion control they expect very close to 100% control and 85% control is not acceptable. It is a benefit that they notice and enjoy however because it was customers that originally told me that the product gave dandelion control. I had not noticed it. I did go out and put on plots, however, and confirmed it. The remaining three pictures marked V, VI and VII are all taken of the Salt Lake Country Club and show the original *Poa annua* plots which I applied. The pictures were taken fairly early in the spring. The winter had been an open winter as is frequently the case in Salt Lake City. During such a winter, the shallow rooted *Poa annua* suffers seriously from drout injury. The deeper rooted perennial bluegrasses are able to obtain enough moisture to grow and so green up earlier. The *Poa annua* is still distinctly yellow from its winter injury and so the dark bluegreen plots of perennial Kentucky bluegrass show up very plainly. Certainly the most interesting plot of the three, which were meant to be three replications is in the left center of the picture. This was the first plot I applied. I was chagrined to find that my spreader was not working properly and that most of the material was coming out of one side of the spreader. I examined the spreader carefully but could not see what the difficulty was. The plot was already ruined and so I continued with the spread while my assistant returned to town to buy a new spreader. The plots in the picture are three years old. The *Poa annua* had not disappeared from the plots completely until the fourth year. The plot in the left middle of the picture, which was not properly applied, proved to be the most interesting plot of the group. As you can see, perennial bluegrass established only where the PAX went and where the PAX did not go the *Poa annua* maintained its hold. Please note how straight the lines of the plots are. There is no tendency for this vigorous growth of perennial Kentucky bluegrass to invade the *Poa annua* under conditions of high phosphate and excessive watering which is characteristic of most golf courses, perennial grasses simply cannot compete with *Poa annua* without chemical help. It is interesting to notice that in the background is the rough. Because the rough is characteristically neglected and not heavily fertilized it has a good growth of perennial grass on it. *Poa annua* is nowhere near as sensitive to arsenical treatment as crabgrass. By the fifth year, the plots are beginning to fail and *Poa annua* is beginning to reinvade. This is shown in the picture marked Exhibit VI. These plots are thirty feet square. I am standing in the middle of a plot that is five years old. The previous fall (the fourth year) the half of the plot on the left hand side of the picture was retreated at half rate (ten pounds per thousand square feet). One can see that on the left the bluegrass is maintaining itself without invasion from the *Poa annua* whereas the right half is beginning to be reinvaded with *Poa annua* after having been free. Of course when a whole fairway is treated and the *Poa annua* is destroyed, it can be expected to hold a considerably longer period than three years simply because there is not a ready source of seed to reseed the areas. But the chemical inhibition from the arsenical begins to fail noticeably by the end of the fourth season when relatively small areas are treated that are surrounded by heavy growth of *Poa annua*. The photograph marked Exhibit VII shows the selectivity of the treatment but it shows an improper use of the material. It is a photograph of a corner of a plot. *Poa annua* is extremely sensitive to arsenical very early in the Spring or late in the Fall. This plot was applied late in the Fall. Grass was still growing vigorously, but the end of the season was approaching. The *Poa annua* died very quickly and bare areas developed. As can be seen, the perennial bluegrass is doing extremely well in the treated area. Nevertheless, the *Poa annua* has died so quickly that the perennial turf is unable to keep up. If an entire fairway was treated in this way, it would most certainly be out of service for the entire season. That is of course what happens when sodium arsenite or some of the other techniques are used to control Poa annua on a fairway.

B. R. Ellison

B. R. Ellison

Subscribed and sworn to before me

this 25th day of April, 1973.

Mary A. Bolliger

Notary Public
Residing at Salt Lake
City, Utah

EXHIBIT I

Excerpts from Southern California Turfgrass Culture, Volume 6, No. 4, October 1956. This publication is the official organ of the Southern Turfgrass Council. This excerpt is a complete article written by Dr. Victor B. Youngner and Tosh Fuchigami of the Department of Floriculture and Ornamental Horticulture, University of California at Los Angeles. The residual control which the two sets of replicated plots of Pax supplied in this experiment resulted in the first entire golf course application with our product. No attempt was made to sell the Bel Air Country Club the product. We did agree to supervise the application.

The product used in these tests was essentially the same as the product we are marketing today except the amount of nitrogen in the product has been reduced from the original 7-0-0 to the present 4-0-0. Also, Heptichlor was substituted for Chlordane as the insecticide.

It is interesting to note that penetration into the subsoil of arsenical is so poor and so slow that it is an ineffective control for white grubs. Vertually no control of white grubs is obtained with an application of any arsenical that season although reasonably good control may be obtained for the following season. To obtain quick control of white grubs and lawn moth larvae one must use a material that penetrates more rapidly than arsenicals.

Southern California Turfgrass Culture

OCTOBER, 1956 VOLUME 6 - NUMBER 4

WEED CONTROL ADJACENT TO GRASSED AREAS

M. H. Kimball[1]
Boysie Day[2]
Chester L. Hemstreet[3]

Turfgrass managers are nearly always responsible for maintenance of flower beds, plantings of trees and shrubs, paths, or small drainage channels, in addition to actual areas of lawns. Control of weeds in such areas, in gravel and flagstone walks, in parking areas and patios adjacent to turf is a serious problem. Machine methods are not adaptable to most situations and chemical methods are often hazardous to adjacent turfgrass and ornamentals. The roots of trees and shrubs are often present, limiting the use of soil-acting chemicals and foliage sprays that leave toxic soil residues.

There are a large number of chemical weed killers commercially available. Some are general weed killers while others control only certain weeds. Some may be used safely in the presence of other plants in specific instances, but the resistance of most ornamental species to the newer herbicides is not known.

Soil Sterilants

When properly applied soil sterilants are not hazardous to nearby turfgrass. Leaching carries the chemicals predominantly downward with very little lateral movement. The principal hazard is to trees and shrubs having roots extending into treated areas. Where distance from trees is considerable -- 30 to 50 feet or more -- it is advantageous and relatively safe to use permanent soil sterilants. Single treatments of the urea herbicides (CMU or DCMU), chlorates, borates, arsenicals, or combinations of these materials control all vegetation for several seasons and may be renewed periodically at relatively low cost. These chemicals are leached into the soil by rainfall or irrigation. They are toxic to all plants and will be picked up by roots and transported to trees and shrubs where systemic injury may result. For long-term sterilization CMU may be used at rates of 20 to 40 pounds per acre, borax or chlorate-borate mixtures at two to four pounds per 100 square feet, and borascu at four to eight pounds per 100 square feet. These chemicals are applied dry or may be dissolved and sprinkled or sprayed on the soil surface. Treated areas should be sprinkler-irrigated (avoiding runoff) to take the chemical into the soil.

Fumigants

Fumigants may often be used to advantage to control perennial weeds near turf plantings. Control by fumigation is only temporary, however, as no toxic materials remain in the soil and recontamination can occur immediately. Fumigation kills the roots of trees and shrubs in treated areas, but this is a "pruning" action, killing the roots contacted. There will be no systemic injury due to absorption of chemicals. If large areas of the root zone are treated, resulting in root killing, the plants may suffer from lack of ability to absorb sufficient water.

Methyl bromide at one pound per 100 square feet applied under well-sealed tarps and held for 24 hours controls such weeds as nutgrass and bermudagrass, and in addition kills most weed seeds. Methyl bromide is very poisonous and must be handled with caution. Injections of two ounces of carbon bisulfide in holes six inches deep and twelve inches apart control deep-rooted perennials such as wild morning glory. The vapors of carbon bisulfide are highly explosive. Ethylene dibromide and DD control nutgrass, oxalis, and other tuberous or bulbous species when injected at the rate of one-half ounce per hole with twelve-inch spacings and four to six inches deep.

Vapam -- a new liquid fumigant -- may be used to control bermudagrass and other perennials. The material is used at the rate of one quart per 100 square feet. To fumigate an area Vapam is mixed with water and sprinkled or sprayed on the soil surface then leached in by sprinkler irrigation. Depth of fumigation may be controlled by the amount of water used before application and to carry the chemical into the soil. To kill the roots and rhizomes of deep-rooted weeds deep leaching is needed. For control of shallow species such as bermudagrass less water is required. Excellent control of shallow-rooted perennials above tree or shrub roots has been obtained by first thoroughly soaking the soil and then while it is fully wet, applying the Vapam. Leach it immediately into the soil by sprinkler irrigation. Apply only enough to carry the material two to four inches deep, normally about 1/4 inch of water.

Results with Vapam are most reliable when the chemical is uniformly diluted in the irrigation water and soaked to the desired depth. One way to do this is to meter the Vapam into the water of a sprinkler system

(CONTINUED NEXT PAGE)

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[1] Extension Ornamental Horticulturist, University of California.

[2] Asst. Plant Physiologist, University of California, Riverside.

[3] Farm Advisor, Agricultural Extension Service, San Bernardino County.

CONTROL OF CRABGRASS WITH CHEMICALS

Victor B. Youngner and Tosh Fuchigami
Department of Floriculture and Ornamental Horticulture
University of California at Los Angeles

Chemical control of crabgrass has been the subject of many investigations in recent years. As a result of this numerous preparations are now available for the control of crabgrass infestations in established turf.

Tests of several crabgrass herbicides were conducted on the 15th Fairway of the Bel-Air Country Club of Los Angeles during the summer of 1956. The area used for the tests had been heavily and uniformly infested with hairy crabgrass, Digitaria sanguinalis, in 1955. The turf in the area consisted of a mixture of bermuda, bluegrass, bents, and fescues. Both pre-emergence and post-emergence materials were used in this test. Seven chemicals, applied at various rates and schedules, and a check plot made a total of 14 treatments as shown in the table. The chemicals used were:

1. Pax AR-76, 8.25% standard lead arsenate, 25.11% arsenous oxide, 0.35% technical chlorodane
2. Alanap 1-F, 1% N-1 naphthyl phthalamic acid
3. Crag Herbicide 1 - 90% 2,4-dichlorophenoxyethyl sulfate
4. PMAS - 10% phenyl mercuric acetate
5. Standard lead arsenate with Milorganite
6. 18.90% disodium methyl arsonate anhydrous
7. Experimental herbicide 140 - 20% Sodium Arsonoacetate.

All treatments were randomized in 4 replications. Each plot was 100 square feet in size.

The first application of pre-emergency materials was made on March 1, 1956, before crabgrass had begun to to germinate. Crabgrass seedlings in the two-leaf stage were first observed March 22, 1956. The first post-emergence herbicides were applied at this time.

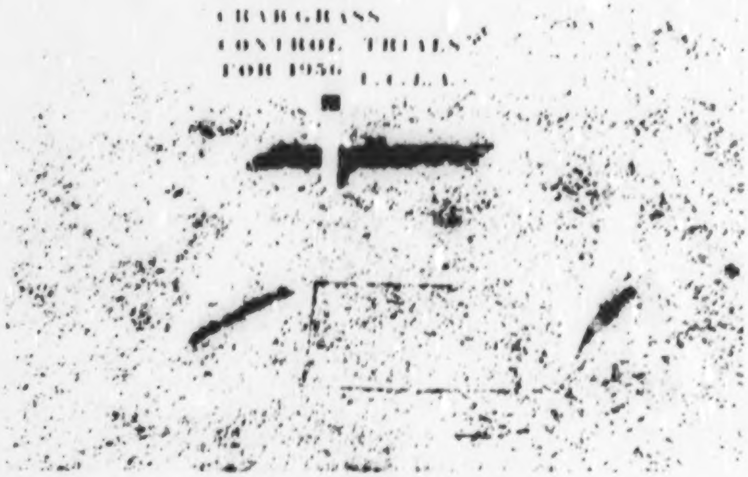
Phenyl mercuric acetate applications were begun when the seedlings were small as observations have shown this to be the most effective period for the use of this material. Disodium methyl arsonate and Experimental herbicide 140 were first applied at the time of peak crabgrass germination. Turf injury and discoloration notes were taken one week after treatments. Estimates of the number of crabgrass plants surviving each treatment were made by counting the number of plants found in four one-square-foot plots taken at random in each treatment. These counts were made twice during the summer, June 18, and September 13.

The results of this experiment are presented in the table below:

CRABGRASS CONTROL IN TURF WITH VARIOUS CHEMICAL TREATMENTS

TREATMENTS RANKED ACCORDING TO EFFECTIVENESS — JUNE 1956 READINGS	APPLICATION DATES	APPLICATION RATE PER 100 SQ. FT.	NUMBER OF CRABGRASS PLANTS PER SQ. FT. AVERAGE OF 4 REPLICATIONS		REMARKS
			JUNE 18, 1956	SEPT. 13, 1956	
1. Pax AR-76. Pre-emergence 1 application	March 1	3.33 lbs.	0.06	0.00	Slight turf injury noted 1 week after treatment. Followed by improved color
2. Pax AR-76 Pre-emergence 1 application	March 1	2.5 lbs.	0.06	0.00	Same as above
3. Disodium methyl arsonate Post-emergence 2 applications	May 14, 23	0.67 oz. in 1 gal. water	0.56	11.25	No turf injury or discoloration
4. Alanap 1-F. Pre-emergence 3 applications	March 1 April 1 May 15	1.8 lbs.	1.06	1.56	No turf injury. Improved color 1 week after treatment
5. PMAS. Post-emergence 3 applications	March 22, 29 April 15	0.25 oz. in 1 gal. water	1.19	10.81	No turf injury or discoloration
6. Alanap 1-F. Pre-emergence 4 applications	March 1, 29 May 14 June 22	1.8 lbs.	1.56	2.50	No turf injury. Improved color 1 week after treatment
7. Alanap 1-F. Pre-emergence 3 applications	March 22 May 14 June 22	1.8 lbs.	1.56	22.31	Same as above

IMPROVED TURF QUALITY
RESULTING FROM
CHEMICAL TREATMENT
TO
DESTROY CRABGRASS →



The crabgrass population estimates as presented in this table show that a number of chemicals are now available which will greatly reduce crabgrass infestations with little or no injury to the desirable turfgrasses. The great turf improvement which was obtained is shown in the accompanying photograph.

Several additional observations should be mentioned. All pre-emergence materials must be applied before any crabgrass seed germinates for best results. In southern California this should be no later than early March. Several applications of post-emergence herbicides must be made to obtain good kill of crabgrass. A second series of post-emergence herbicide treatments should be made in late summer to kill new crabgrass plants from late germinating seed. Improved turfgrass color and growth was obtained from the Pax and Alanap 1-F in addition to an excellent control of crabgrass.

In addition to this series of tests a small test of Du-Pont Crabgrass and Chickweed Preventer (Neburon) was made in an adjacent area. This material arrived too late to be included in the regular test. Unfortunately, by this time crabgrass seed had begun germination. Nevertheless population counts showed this material to be an effective herbicide for crabgrass. It was applied in a single application at the rate of 0.8 oz. for 100 square feet of turf.

8. Crag Herbicide 1. Pre-emergence 4 applications	March 1, 29 May 14 May 23	0.30 oz. in 1 gal. water	2.31	11.06	No turf injury or discoloration
9. Standard lead arsenate + Milorganite. Pre-emergence - 1 application	March 1	11 oz. lead arsenate + 4 lb lbs. Milorganite	4.50	17.13	No turf injury or discoloration. Improved color
10. Disodium methyl arsonate Post-emergence 3 applications	May 14, 23 June 22	0.67 oz. in 1 gal. water	5.44	20.50	No turf injury
11. Crag Herbicide 1. Pre-emergence 4 applications	March 29 May 14, 23 June 22	0.30 oz. in 1 gal. water	11.00	32.81	Same as above
12. Experimental Herbicide 1. Post-emergence 5 applications	May 14, 23 June 1, 7, 22	0.5 oz. in 1 gal. water	12.25	45.31	Same as above
13. Disodium methyl arsonate Post-emergence 1 application	May 14	0.67 oz. in 1 gal. water	12.31	65.44	Same as above
14. Check. No treatment			23.88	95.94	
L.S.D. at Probability of 5%			3.86	32.90	

A difference between treatments greater than 3.86 for the June counts and 32.90 for the September counts is significant at 5% Probability level.

EXHIBIT II

The "Rocky Mountain Golf Course Superintendent's Reporter", Volume 6, No. 6, published June 1971. This publication is the official organ of the Denver Chapter of the Golf Course Superintendents Association of America. It is an article by Ben Struemps, Superintendent of the Green Gables Country Club in Denver. He describes how he converted his fairways from a turf containing approximately 80% Poa annua to a turf containing less than 10% with the use of Pax Three Year Crabgrass Control. I personally supervised the application. It was extremely successful and as a result several other country clubs in the Denver area have treated with Pax.

THE
ROCKY MOUNTAIN
GOLF COURSE
SUPERINTENDENTS
REPORTER

Volume 6, Number 6 June, 1971

POA ANNUA CONTROL

By BEN STRUEMPF

In April, 1970, as the new superintendent at Green Gables Country Club, Denver, Colorado, I was informed that the Club's most trying problem was the annual loss of turf due to *Poa* wilt.

With approximately 80% *Poa* in fairways, tees, and greens the first problem was selecting the best course of action for our Club. One method would be to disregard the health of existing blue grass and maintain for *Poa*. After looking at the merits of this method, we looked for a better method to cure the summer wilt, since *Poa* is a fair weather friend, more susceptible to disease and having a hard winter will kill it as easily as heat and drought. Another possibility was to use sodium arsenite and burn off three fairways a year and renovate completely. With the knowledge that the members would never accept this method, we decided on the one remaining alternative which was to remove the *Poa* and leave the existing perennial bluegrass. After reasoning this far, we still had other considerations as to the type of product to use for this course. Growth retardants and seed head preventatives were ruled out as too slow for fairways and not safe for greens at present. Pre-emergence chemicals were decided by our committee as too slow for fairways but the only safe means for greens. Tricalcalcium arsenate was disregarded as too fast with the large amount of *Poa* that we had. Our final decision was to use an intermediate product: Pax three year compound of lead arsenate and arsenical oxides. This product offered a slow post emergence control (one to two years to kill existing *Poa* and three years of pre-emergence control).

Our June Host, Ben Struempf, illustrates where 100% Poa Annua thrived last year. The area has almost completely filled in with bluegrass through Ben's efforts.

Our program ran as follows: On April 15, 1970, at #18, we started over-seeding with a blue grass mix (10% Windsor, 15% Merion, 15% Fylking, 15% Delta, 15% Newport, 15% Park and 15% Common) and aerifying all fairways on the course. On May 18, we started our Pax treatment. Note that the time of application is important to achieve the desired results. An early application on lush growing *Poa* or a late appication in hot weather will give a more rapid kill. Ideal timing for a slow kill is to apply just after seeds heads have formed. Pax was applied at half rate one day, watered in, and the other half the following day for uniformity. A Scott's drop spreader was used for spreading. We dropped phosphates from our fertilizer program in 1970, and will keep it out in the future as our soil test averages 2400 lbs available phosphate per acre.

On September 21, we started our second over-seed of the course. As a newcomer to Denver, I made a mistake in timing both over-seedings. The April 15 seeding was early and did not germinate until mid-July and the fall seeding was late and did poorly. I chalk this up to experience. The September over-seed was followed with an aerification. In August 1970, we lost *Poa* in some large areas, primarily on green approaches. The areas that wilted, wilted wet, and the only logical reason for this was the excess of thatch. The thatch, being so thick that the arsenicals had no soil particles to attach to, caused the arsenicals to become gaseous more rapidly and consequently, give a more rapid kill on the *Poa*. (Water was never withheld.) This did not discourage me, as the areas that wilted were not fit to grow grass. With 2 to 3" of thatch and stratification of sand and peat, it was an excellent opportunity to renovate, grade properly and re-sod. Fairways were cut at 1¼" through 1970.

1971 is a good year for Green Gables Country Club. The *Poa* has been reduced to approximately 10%, and the blue grass is dense and healthy. We are mowing fairways daily at 1⅛".

In mid-June, we still have small bare areas which are filling in rapidly. We over-seeded fairways the first of June this year, which is coming well. We will overseed fairways again in August. We have been using a Rogers Mod. 58 verticut with the seeder attachment for our overseeding. We are aerifying once a month. By 1972, we expect solid blue grass fairways that will stand a ball up when cut at 1⅛". Little has been said about the greens and their 80% plus or minus *Poa*. Pre-emergence type control has been decided on for greens but withheld until fall of 1972 to allow time to improve the quality of the bent grass stand. Finding out that the front nine was old German seeded bent, the back nine Seaside bent, and the two new greens Pencross, we decided to head all greens in the direction of Pencross by overseeding.

We overseeded all greens three times in 1970 with Pencross and will repeat in 1971. Our intent is to introduce enough Pencross that in years to come, it will dominate and give more uniform greens. In fall, 1971, we will start our pre-emergence program on greens with a Benzenesulfonamide type product until results area chieved.

EXHIBIT III

Superintendents of prestige country clubs in the Beverly Hills and Bel Air area of Los Angeles. All of these clubs are exceptionally wealthy clubs and beautifully maintained. Brentwood Country Club and Hillcrest Country Club treated their entire golf course after seeing the excellent results obtained from the treatment at Bel Air Country Club.

there's nothing like PAX for golf course crabgrass control that lasts and lasts and lasts!

Here's what these experts say:

MAJOR FRED BOVE, Superintendent of Brentwood Country Club, Brentwood, California says: "Two years ago we decided to treat the entire 18 fairways with PAX after viewing the years of residual control which PAX demonstrated at several top country clubs in Southern California. And now the overall success of Pax exceeds 99% in elimination of existing crabgrass. Not only that, there was a big reduction of annual weeds and the nitrogen shot in the arm was a welcome asset to the application."

JOE MARTINEZ, Superintendent of the Bel Air Country Club, Bel Air California says: "You don't have to tell me anything about PAX. I found it so effective that I have incorporated it into my maintenance program. I use it annually for spot treating and re-treating tees and aprons as necessary. I went through three years of almost total crabgrass control after just one application of PAX to 16 fairways. That initial expenditure was extremely worthwhile and is all it took to convince me."

CHARLES (CHUCK) PRIDAY, Superintendent of Hillcrest Country Club, Los Angeles, California says: "I've been at Hillcrest for 36 years and have been waiting for something like PAX all that time. I decided to make an 18-fairway application of PAX in March, 1962. The crabgrass control so far has been excellent. I'd say it's been a good 98% kill."

That's what the experts think and say about PAX

PAX is the ideal crabgrass control for golf courses. It's simple and easy to apply on a large-scale basis and the results are extremely effective.

You can cover an entire 18 hole golf course in just 3½ days using just five men and two tractor towed spreading machines.

WRITE TODAY FOR MORE INFORMATION:

Pax Company Dept. 100
580 West 13th South, Salt Lake City 15, Utah

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EXHIBIT IV

At the time of Dr. Frost's visit, he wanted to see a plot that had been treated with Pax Three Year Crabgrass Control which encouraged the growth of desirable grass species as mentioned in the thesis by Richard Stadther. I took him to the plot shown in the photograph. This plot was applied by the Extension Agronomist, Lewis Jensen, of the Utah State University. It was applied in Liberty Park in Salt Lake City. I had nothing to do with the application. It was applied at standard rates. I am photographed on the plot four years after its application pointing to the extremely sharp line of crabgrass control which this product gives. Plots on each side of the Pax plot are treated with various other crabgrass control materials, but have long since lost any weed controlling abilities which they had. The plot is not ten years old and is still plainly discernable. Dr. Frost was able to see it, even though crabgrass has not germinated in our area. He was able to discern it because of the more desirable turfgrass species which were growing on the plot than in the contiguous turf areas.

EXHIBIT V

Dr. Frost ask to be taken to a golf course that had treated its fairways with Pax. I took him to two. Unfortunately our season is very late and the turf is just breaking dormancy. Both country clubs which we went to, in fact all country clubs in our area, were treated with Pax Three Year Crabgrass Control so long ago that they are now in the process of retreating.

Three plots are shown in the photograph. At the time of the photographs, the plots were four years old. The plot in the middle left of the picture is probably the most interesting of the plots because the spreader was not working properly at the time that it was applied. Most of the material came out of one side of the spreader with the result that some areas were overtreated and other areas received no treatment at all.

Perennial bluegrass filled in the treated areas but was never able to compete with the Poa annua which did not receive the treatment.

The photograph was made fairly early in the season. When Salt Lake has an open winter, Poa annua suffers rather severly from drout injury because it is shallow rooted. This was an open winter and the Poa annua is showing the drout injury. The perennial bluegrass which has come back is deep rooted and therefore is not showing winter drout injury. That is the principle reason why it has greened up and is growing so much earlier.

No attempt at reseeding was made although in this area actual counts in the turf showed that the perennial Kentucky bluegrass plants were running approximately five plants per square foot. At no time was this turf left bare. The Poa annua went out slowly and the bluegrass filled in as the Poa annua went out.

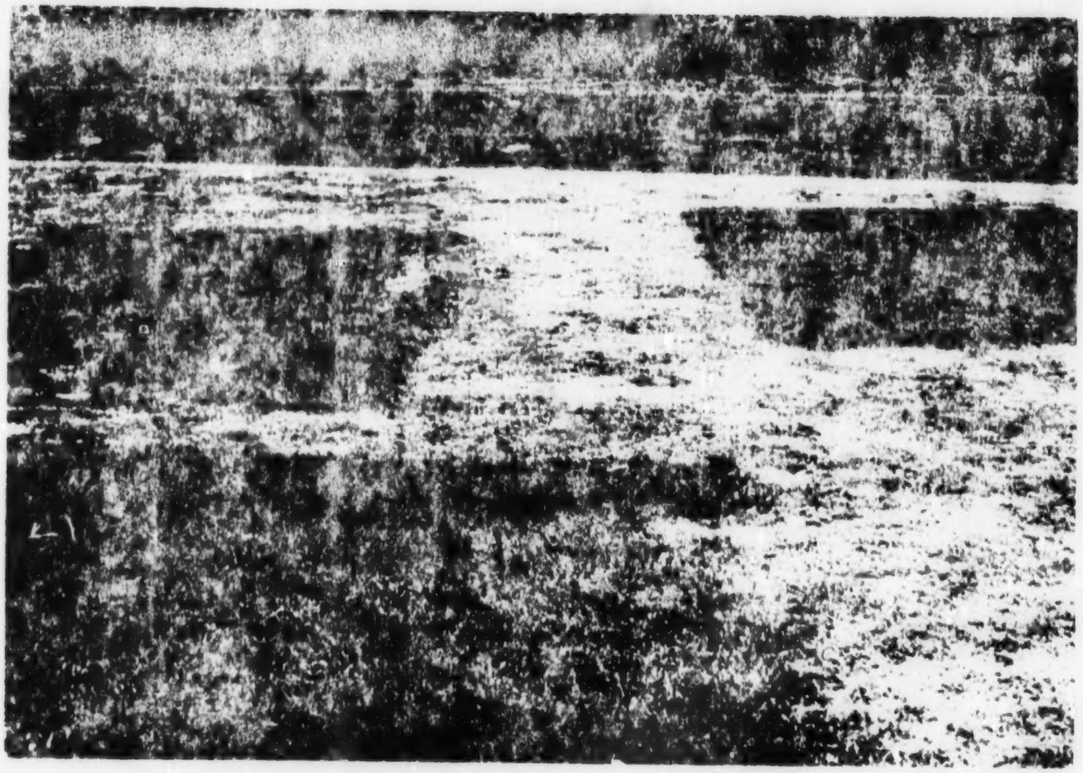

EXHIBIT VI

In the early Fall of the fourth year of the original application of the plots, I retreated the left hand half of one plot at half rate or ten pounds per thousand square feet of the product. The photograph shows me standing at mid-line of that plot. These plots are thirty feet by thirty feet. The untreated half on the right is beginning to fail and one can see that *Poa annua* is beginning to reinvade the untreated area. This shows that even a vigorous perennial bluegrass turf cannot compete with *Poa annua* without chemical help on the average golf course fairway.

EXHIBIT VII

This photograph shows the selectivity of the product but nevertheless the product has been misused in this area. This shows a corner of a large plot. Poa annua is extremely sensitive to arsenicals early in the Spring and late in the Fall. If the application is made during these times, the Poa annua dies very rapidly leaving a heavily infested area virtually bare. Such a treatment is not practical on a heavily infested fairway. The desirable thing is to avoid killing the Poa annua and merely shift the factors of competion in favor of the deeper rooted perennial bluegrass. This is done because the major effect of arsenicals in the soil is to restrict the division of the cells of the root meristems. As the result of this, the roots do not grow and form root hairs. Since the root hairs are the major site of uptake of both minerals and water, the Poa annua, with its shallow root system, is maintained under almost constant moisture and nutritional stress. Under these conditions, the perennial bluegrass of whatever variety, is able to compete successfully with the Poa annua and replace it. This is the only way I know to treat a heavily infested fairway without leaving bare areas or making the fairway unplayable for the balance of the season.

[redacted]

17 Rosa Road
Schenectady, N.Y. 12308
April 6, 1973

Writing on behalf of the EPA PAX Company Arsenic Advisory Committee, may I seek your help and advice? PAX Three-year Crabgrass Control (25.11 % arsenic trioxide with 8.25 % lead arsenate), although not now in use in New York, is permitted for use on home lawns as well as golf courses in some western states. Our assignment is to assess the possible hazard from this product in particular, but with some view to related products such as calcium arsenate and lead arsenate for turf improvement.

Can you provide instances in which the use of such products has caused measurable toxicity or other adverse effects in animals or humans? Thus far, we have been unable to find substantial evidence for such poisoning.

According to the labels, grass clippings after such treatment are not to be fed. At least one poisoning from accidental feeding of PAXXtreated clippings was reported to have led to death in horses. One would think that clippings from treated golf courses would have caused similar problems. Have you heard of any such? Do you know of any proven hazard to any species from turf improvement arsenical products?

If you can possibly do so, please respond promptly. Our deadline comes soon.

Thanks for your help.

Yours sincerely,

Douglas V. Frost, Chairman
PAX Company Arsenic Advisory Committee

Reproduced from best available copy.

Midwest Regional Turf Foundation

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WILLIAM H. DANIEL
Department of Agronomy
Purdue University
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Room 2-303, Lilly Hall
Phone 317-49-43195

April 10, 1973

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EX OFFICIO
LEE RECORD
U.S.G.A. Green Section
Crystal Lake, Illinois
60014

Dr. Douglas V. Frost
17 Rosa Road
Schenectady,New York 12308

Dear Dr. Frost:

I appreciate getting your inquiry of April 6 concerning arsenic on turf areas.

I have worked with arsenicals since first coming to Purdue in 1950, have hundreds of kodachrome slides showing the results of using arsenicals to control crabgrass, goosegrass, chickweed, etc. Have written numerous articles recommending and proposing its use for turf managers on professionaly managed turf. And, I currently believe that the granular formulations, such as Chip-Cal 49% tri-calcium arsenate should continue to be permitted for use on professionally managed turf areas.

I recall about 1958 when the PAX Company had all the pre-emergent market in Indiana. They sold eight car loads of material in the state that year. By 1965 they had withdrawn their sales in Indiana because other products were preferred by homeowners and were safer for homeowners.

I recall Vaughan Seed Company selling one million pounds of granular Pre-Kill to homeowners about 1960-61. They had two complaints - one a refund of money, one a replacement of product, but they also stopped production for homeowners.

The Farm Bureau of Indianapolis sold Stopps. This also was changed to a less toxic form - all before EPA came along.

I have every reason to encourage EPA to continue to allow professional turf managers to use arsenics on professionally managed turf areas, such as golf courses. We have no companies that are now recommending arsenicals for home lawns, or for novice use. The Chipman Company is the only one surviving out of eight that used to sell calcium arsenate. They have a good educational program and two professional turf managers.

Note the enclosed leaflet. They should be encouraged to stay in business for if they go we will lose a Poa annua control program now in practice on over 1,000 golf courses having achieved Poa annua removal and there are at least another 1,000 in the process of removing Poa annua with arsenicals. I am not for Chipman, but I am interested in having Poa annua-free golf courses, and feel our society needs good turf for its leisure time and recreational use. There is no glory in having Poa annua on a golf course when citizens need the outdoor recreation.

Know you will have trouble finding cases of arsenic damage to man and animals. The literature says - the body will excrete arsenic, and the history of the workers in arsenic mining and in chemical manufacture shows no cases of excessive damage even to workmen in the plant.

My personal experience of more than twenty years working intimately with golf courses across the United States is that we have no evidence of damage to animals, nor continued damage to persons. Sure, there have been a couple of mistakes where a man got a temporary response. For example, one man quit smoking after unwisely using excessive powdery forms of calcium arsenate years ago before he could get the granular formulation. But, that same man still uses granular forms now. We no longer recommend powdery formulations, although they could be purchased at half the cost per acre treatment.

Good luck on your research towards finding evidence of toxicity! The beauty of the program is it is so widely successful and such limited damage that the program stands as one of those that should be kept.

Therefore, I strongly favor the continued permit to use calcium arsenate in a granular formulation by professional turf managers on professional turf areas as a good procedure which deserves EPA concurrence, and has current, adequate education and research behind it.

Trust this is helpful.

Cordially
Yours for Better Turf,

William H. Daniel
Turf Research & Extension

WHD:kh
Enc. Chip-Cal leaflet

UNIVERSITY OF RHODE ISLAND
KINGSTON • R. I. 02881

College of Resource Development • Department of Plant and Soil Science

April 10, 1973

Mr. Douglas V. Frost, Chairman,
PAX Company Arsenic Advisory Committee,
17 Rosa Road,
Schenectady, N.Y. 12308

Dear Mr. Frost:

I have worked with arsenicals of many kinds in the herbicidal field since the late 1940's. I have never personally encountered, nor have I had first-hand knowledge of, any accidental poisoning as a result of the use of arsenicals.

I do recommend inorganic arsenicals, such as tricalcium arsenate, regularly for weed control purposes and don't feel that there is currently a substitute for certain purposes.

I hope this information is helpful to you.

Sincerely,

C.R. Skogley
Professor of Agronomy
Plant & Soil Science

CRS:lp

COOPERATIVE EXTENSION NEW YORK STATE
Cornell University • State University of New York • U.S. Department of Agriculture
Chemicals-Pesticides Program (607) 256-3283
Caldwell Hall, Ithaca, N. Y., 14850

April 11, 1973

Douglas V. Frost, Chairman
PAX Company Arsenic Advisory Committee
17 Rosa Road
Schenectady, NY 12308

Dear Mr. Frost:

I have your letter of April 6 concerning possible hazards of arsenic on turf. Possibly you are famliar with the fact that calcium arsenate can be used in New York State on turf for the control of poa annua in prescription programs with a "B" permit. This material was originally excluded from use but was later allowed under prescription type programs primarily because no substantial evidence of injury to the environment or animals in the environment could be found. We were unaware of any problem resulting from the feeding of golf course grass clippings to animals. In the event such were the case, it would seem logical that a restriction on such use might be employed as is the case on several of our agricultural commodities.

I am sorry that I am unable to come up with anything that would be helpful to you.

Very truly yours,

James E. Dewey
Extension Program Leader
Chemicals - Pesticides

JED/gmw

CHEMICALS • PESTICIDES
NEW YORK PROGRAM

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RUTGERS UNIVERSITY *The State University of New Jersey*

COLLEGE OF AGRICULTURE AND ENVIRONMENTAL SCIENCE
P. O. Box 231
Department of Soils and Crops
New Brunswick, New Jersey 08903
(201) 247-1766 Ext. 1427

April 12, 1973

Dr. Douglas V. Frost
17 Rosa Road
Schenectady, New York 12308

Dear Dr. Frost:

In answer to your request on the dangers of arsenate treatments such as "Pax", I have had no direct association or knowledge of injury to animals or humans from use of these chemicals (Ca arsenate, lead arsenate, and "Pax") on turf during my 25 years in my position.

Since there is great concern about keeping animals away from arsenic-treated turf and most of the use has occurred on golf courses where grazing animals are scarce, use of arsenates on turf has not created ready occasion for such incidents.

It is my understanding that New Jersey is currently considering chemical bans, restricted use and licensed use. I have no idea where arsenates will fall in this program.

The arsenates are the only efficient type of herbicide for control of annual bluegrass in closely mowed turf such as occurs on golf courses of the Northeast. However, I have no interest in this type product for home lawns because of the hazard and the fact that annual bluegrass and crabgrass can be controlled by means other than arsenates.

I trust these comments are helpful.

Sincerely yours,

Ralph Engel

Ralph E. Engel
Research Professor in
Turfgrass Management

Nelson [illegible], [illegible] 26:105, 1972 :
Mortality among orchard [illegible] exposed [illegible]
[illegible] cohort study.

jc

University of Toronto
TORONTO 181, CANADA

DEPARTMENT OF BOTANY
Tel. 416-928-3534

Chas Lears - Please return to me
I want to look up his papers. D.V.F.

April 23, 1973

Dr. Douglas V. Frost,
Chairman,
EPA PAX Company Arsenic Advisory Committee,
17 Rosa Road,
Schenectady, New York 12308

Dear Sir:

Regarding your inquiry for information on environmental contamination by heavy metals. My doctorate study involved an assessment of metal fallout from a nickel refinery and a zinc-lead smelter and subsequent accumulation in vegetation.

Associated with the zinc-lead smelter I found elevated levels of Zn, Pb, Se, As, Sb, Cd and Cu.

Associated with the nickel refinery I found elevated levels of Ni, Cu, Co, Se and In.

This data may be obtained from my doctoral thesis:-

Roberts, T. M. (1972) The Spread and Accumulation in the Environment of Toxic, Non-ferrous Metals from Urban-Industrial Sources. Ph.D. Thesis Wales

Other publications relevant to the Swansea study include:-

Goodman, G. T. and Roberts, T. M. (1971) Plants and soils as indicators of metals in the air. Nature (Lond.) 231, pp. 287-292.

Roberts, T. M. (1972) Plants as monitors of airborne metal pollution (a review). J. Environmental Planning. vol. 1 no. 1, pp. 43-54.

An indication of atmospheric metal levels 1½km downwind of the nickel refinery can be obtained by looking at the data for the "Trebanos site" on page 255 of Nature, vol. 241 No. 5387, January 26, 1973 (Trace elements in the atmospheric environment. Peirson, D. M. et al.)

Continued.....

I hope that this information will be of use. I would be grateful for any reprints of your study.

Yours sincerely,

Mike Roberts

T. M. Roberts,
Lecturer

TMR/ad

UNITED STATES DEPARTMENT OF AGRICULTURE
AGRICULTURAL RESEARCH SERVICE

NORTHEASTERN REGION
AGRICULTURAL RESEARCH CENTER
Beltsville, Maryland 20705

May 7, 1973

Subject: EPA PAX Co. Arsenic Advisory Committee

To: Douglas V. Frost
Consultant, Nutrition Biochemistry
17 Rosa Road
Schenectady, New York 12308

In response to your letter of April 6, 1973, we know of no instances where PAX has caused toxicity problems when used in the recommended manner. Likewise, we know of no instances where any turf improvement product has caused toxic problems when used as directed. It is our opinion that the PAX product, containing 25.11% As_2O_3 with 8.25% lead arsenate for crabgrass control, should probably be allowed to be used. As with any economic poison, there is always the possibility of misuse by people, but I don't think we should regulate the ability of citizens to use a product safely.

The lead inclusion in this product does not seem to be great enough to prevent the use of PAX on turf. The placement of warning statements in a more prominent position on the label may help in preventing accidental poisonings, particularly from grass clippings. Other than that, if the pesticide is watered in, like it's supposed to be, we can see no further problems with the material.

E A Woolson

E. A. Woolson
Analytical Chemist
Pesticide Degradation Laboratory
Agricultural Environmental
Quality Institute

Philip C Kearney

Philip C. Kearney
Leader
Pesticide Degradation Laboratory

COOPERATIVE EXTENSION NEW YORK STATE
Cornell University • State University of New York • U.S. Department of Agriculture
Chemicals-Pesticides Program (607) 256-3283
Caldwell Hall, Ithaca, N. Y., 14850

May 8, 1973

Douglas V. Frost, Chairman
PAX Company Arsenic Advisory Committee (EPA)
17 Rosa Road
Schenectady, NY 12308

Dear Mr. Frost:

I have your letter of April 14 and regret that I have delayed so long in my response. However, I tried to determine whether or not there were any restrictions specifically listed for materials which might be used on turf. I was unable to find any restrictions for such uses.

Restrictions and limitations are quite common on many of our pesticides and particularly for those that might be used on forage crops or for use on crops which sometimes might be used as livestock feed.

It would not seem out of order to me for such restrictions to be placed on products which are used for the treatment of turf. We have had an occasional inquiry requesting information about the possible use of grass clippings from sod farms as a possible livestock feed. However, because of the nature of the compounds being used, chlorinated hydrocarbons like DDT, dieldrin, and chlordane, we warn them against this practice due to the accumulation in the animal even when extremely small residues might be present. We have had no inquiries that I can recall from golf courses, but if this is a possibility then a warning against such practice might be included as part of the label.

Very truly yours,

James E. Dewey
Extension Program Leader
Chemicals - Pesticides

JED/gmw

CHEMICALS • PESTICIDES
NEW YORK PROGRAM

New York State College of Agriculture and Life Sciences, New York State College of Human Ecology, and New York State Veterinary College at Cornell University, Cooperative Extension Associations, County Governing Bodies, and United States Department of Agriculture, cooperating

April 6, 1973

Mr. George J. Butler
The Western Area Occupational
Health Laboratory
N.I.O.S.H.
Post Office Box 8137
Salt Lake City, Utah 84108

Dear Mr. Butler:

Mrs. Fran Brogan of the Cincinnati office referred me to you concerning a question dealing with the use of arsenic in Salt Lake City. I am currently serving on an E.P.A. advisory committee having to do with the use of a product called PAX Crab Grass Control. This material is made in Salt Lake City and is used in considerable quantity in that area. Are you aware of any complaints concerning the use of this material for crab grass control in residential areas? The company has provided us with considerable information, but I thought it a good idea to contact the Salt Lake City office simply to learn whether any complaints had filtered into the N.I.O.S.H. office at Salt Lake.

By way of introduction, many of my years were spent in the Division of Occupational Health and, thus, I am calling upon you for privileged information in case such is at hand. There will be no need for you to contact the company, as they have provided us with considerable data so far.

Thanking you in advance for any effort this request may cause.

Sincerely,

Donald J. Birmingham, M.D.
Professor

DJB vp

DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
PHS, HSMHA
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH
WESTERN AREA OCCUPATIONAL HEALTH LABORATORY
P. O. BOX 8137
SALT LAKE CITY, UTAH 84108

April 11, 1973

Donald J. Birmingham, M.D.
Professor
Wayne State University
Gordon H. Scott Hall
of Basic Medical Sciences
540 East Canfield Avenue
Detroit, Michigan 48201

Dear Dr. Birmingham:

Your introduction was unnecessary since your name is quite familiar to me from your past association with the Public Health Service and your contributions to the field of dermatology.

I, like many other amateur gardeners here in the Salt Lake Valley, am aware of PAX Crab Grass Control, but I have never personally experienced any problems with the use of the chemical. I have also asked some of the staff at this facility who also use the material but there replies were as negative as mine. In addition, I contacted Salt Lake County and Utah State Health Department representatives to see if they had any record of complaints about PAX products, but neither one of the agencies had knowledge of any problems attributed to its use in the community.

I hope that this information will be of some use to you and if I can be of any further assistance, please feel free to contact me.

Sincerely yours,

G. J. Butler
George J. Butler
Sr. Sanitary Engineer
Coordinator, DTS/SLC

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PERSONS WHO APPEARED BEFORE THE COMMITTEE:

Mr. W.B Robins, Manager, The PAX Company.
Mr Donald B. Holbrook, Jones, Waldo, Holbrook and McDonough, Attorneys and Counselors, Salt Lake City, Utah.
Alan K. Done, M.D., Pediatrician and Toxicologist, Medical Division, Food and Drug Administration, Washington, D.C.
E.A. Woolson, Ph.D., Analytical Chemist, Pesticide Degradation Laboratory, Agricultural Environmental Institute, USDA, Beltsville, Md.
William Buck, D.V.M., Professor, Toxicology Section, Veterinary Diagnostic Laboratory, Iowa State University, Ames, Iowa.

PERSONS WHO RESPONDED TO LETTERS FROM COMMITTEE MEMBERS:

George J. Butler, Sr. Sanitary Engineer, Western Area Occupational Health Laboratory, NIOSH, PHS, HEW, Salt Lake City.
Bernard Ellison, Research Director, PAX Company, Salt Lake City.
William H. Daniel, Ph.D., Professor, Turf Research, Dept. of Agronomy, Perdue University, Lafayette, Indiana.
Ralph E. Engl, Ph.D., Research Professor, Trufgrass Management, College of Agriculture and Environmental Science, Rutgers University, New Brunswick, N.J.
C.R. Skogley, Ph.D., Professor, Agronomy, University of Rhode Island, Kingston, R.I.

William B. Buck, see above.

Douglas I. Hammer, M.D., Human Studies Laboratory, Division of Health Effects Research, National Environmental Research Center, EPA, Research Triangle Park, N.C.
John V. Lagerwerff, Ph.D., Soils Scientist, USDA-ARS, Beltsville, Md.
Philip G. Kearney, Ph.D. and E.A. Woolson, Ph.D., Agricultural Environmental Quality Institute, USDA-ARS, Beltsville, Md.
M. Norman Anderson, Amax Lead and Zinc, Inc., Boss, Mo.

T.M. Roberts, Ph.D., Institute of Environmental Sciences and Engineering, Univ. of Toronto, Toronto, Canada.
Glen E. Gordon, Dept of Chemistry, University of Maryland, College Park, Md.

James E. Dewey, Entomologist, Chemical Pesticides Program, Caldwell Hall, Cornell University, Ithaca, N.Y.
Walter Durniak, Cornell University Cooperative Extension Agent, Schenectady County, N.Y